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Symposium: Review of Gas and Volatile Agents in Anesthesia

Comparison of Chloroform, Vinethene, and Ethyl Chloride

ROBERT FIDLER, D.O.
Seattle

This paper deals with three of the volatile agents used in general anesthesia, namely Vinethene, chloroform, and ethyl chloride. Their pharmacology, uses, indications, and contraindications will be discussed. There are times, conditions, and situations when the anesthetist may, of necessity, be called upon to use any of these agents, and, at such times, they may be used to excellent advantage.

Anesthesia of various types had been attempted for centuries, but it was not until the early nineteenth century that dependable means began to be developed. In 1831 Soubeiran in France discovered chloroform, and a few years later Simpson in Edinburgh introduced it into the practice of surgery.[1] In 1847 Flourens noted the anesthetic qualities of ethyl chloride, and, although it was used by Heyfelder in the following year, it was abandoned until 1895.[2] Later, in 1930, Leake[3] introduced Vinethene. Although all three agents, chloroform, Vinethene, and ethyl chloride, have some disadvantages, they also have certain advantages and, in the hands of the competent anesthetist, have a place in general anesthesia.

Vinethene, chloroform, and ethyl chloride exhibit certain similar qualities. They are rapidly acting, volatile liquids, mildly unpleasant in induction. They cause an increase in mucous secretions during induction, are moderately irritating to the respiratory tract, have an effect on respiration similar to that of ether, and have a rate of recovery more rapid than that of ether. Chloroform and ethyl chloride are quite stable in suitable containers while Vinethene is unstable after its container is opened. Chloroform with adequate oxygen gives excellent muscular relaxation, Vinethene good muscular relaxation, and ethyl chloride only fair relaxation. Respiratory arrest occurring in deep anesthesia with chloroform or ethyl chloride is due to cardiac toxicity and oxygen want. Cardiac toxicity is undetermined in respiratory arrest with Vinethene anesthesia, arrest more likely being due only to oxygen want. Both chloroform and ethyl chloride commonly cause some arrhythmia. Some degree of liver damage is common with chloroform and may be severe in the presence of hypoxia or pre-existing liver disease. The extent of liver damage from ethyl chloride is undetermined, and it is minimal with Vinethene provided adequate oxygenation is maintained and the procedure is of short duration.

VINETHENE

Vinethene is divinyl ether plus 3.5 per cent of absolute alcohol and 0.01 per cent of phenylalphanaphthylamine. It is also called vinyl ether or divinyl ether. It was first introduced in 1930 by C. D. Leake.

It is a highly volatile inflammable liquid which has an ethereal nonpungent odor and a specific gravity of 2.2.[3] It is readily decomposed by light, heat, and air, forming peroxide and aldehyde. Decomposition is inhibited by ammonia and amines. Administration of this agent can be carried out by open drop, semiclosed, and closed methods, with or without the carbon dioxide absorption technic. Vinethene is easily inhaled and quickly produces unconsciousness and surgical anesthesia. Analgesia is obtained with a concentration of 2 per cent by volume in the alveolar air, anesthesia with approximately 4 per cent by volume, and respiratory failure with 8 to 10 per cent by volume in the alveoli. In order to promote smooth anesthesia Vinethene must be dropped almost continuously during induction and maintenance when the open drop method is used. It has been suggested that the tip of the dropper be kept 1 inch from the mask to decrease the loss of Vinethene by evaporation. The agent is readily absorbed in the lungs and is unaltered in the system. It is also quickly eliminated, and recovery generally occurs in from 3 to 5 minutes. Small amounts are recoverable in the urine and perspiration.

Physiologically the drug causes copious secretion, increases the respiratory amplitude, the heart rate, and the cardiac output.[3] It causes a peripheral circulatory dilatation, and arrhythmia is not uncommon, particularly during induction. Its effect on the liver is similar to that of chloroform but usually not so severe. Central lobular necrosis can be prevented or greatly reduced with the addition of a high concentration of oxygen. A diet high in carbohydrates will also protect the liver from Vinethene damage.

Care should be taken to protect the patient's face from anesthetic burns, and the usual precautionary methods for explosive hazards should be given due consideration. Anesthesia should not be induced until all preparations for surgery have been made as the period of anesthesia should be as short as possible. This drug should not be administered too rapidly or in too high a concentration, as this increases salivation and secretion of mucus and also increases the possibility of overdosage.

When Vinethene is employed anoxia or airway obstruction should not be tolerated under any circumstances, for anoxia enhances the possibility of liver damage. A suction apparatus for the prevention of aspiration of saliva and mucus should be at hand. Preanesthetic medication with morphine and atropine or morphine and scopolamine is preferred. A small dose of one of the barbituric acid derivatives will decrease preanesthetic apprehension. Vinethene can be administered with every type of standard anesthetic apparatus. Administration by the open drop technic at a rate of 60 to 80 drops per minute for induction and 30 to 40 drops per minute for maintenance is advised. Analgesia is usually obtained in 60 seconds or

slightly longer. The patient passes through the second stage very rapidly with some excitement. The third stage of surgical anesthesia elicits the same signs as ether anesthesia. Ether sequence is accomplished by adding ether to the mask. Complete recovery from deep Vinethene anesthesia rarely takes more than a few minutes. The usual treatment for overdosage is discontinuance of the drug, artificial respiration, and administration of oxygen.

The indications for the use of Vinethene are as a preliminary agent to shorten the first and second stage of ether anesthesia; as anesthesia for brief minor surgical procedures, such as dental extractions, reductions of fractures, and obstetrics; and as a complemental agent to nitrous oxide, ethylene, or other inhalation anesthesia. Vinethene should not be used in procedures requiring the use of a cautery or similar apparatus, in the presence of acute upper respiratory infections, for surgical procedures lasting longer than 30 minutes, or for procedures requiring muscle relaxation. Its use is also contraindicated in the presence of hepatic or renal insufficiencies or diseases.

CHLOROFORM

Chloroform is a clear, colorless, volatile liquid; it has a characteristic ethereal odor and a burning sweet taste; it is noninflammable, easily inhaled, and acts rapidly. It is decomposed by light and air and therefore should be kept in colored, well-stopped bottles. It boils at approximately 140 F. U.S.P. chloroform contains a trace of alcohol to convert any phosgene to harmless diethyl carbonate. It is the most potent anesthetic agent available. Chloroform may be administered by the open drop, semiclosed, or closed methods. The technic most commonly employed, the simplest, and the safest is the open method. The closed technic allows rebreathing with a high oxygen concentration, but it is recommended only in the hands of the experienced anesthetist.

Analgesia is obtained with less than 1 per cent by volume in the alveoli, anesthesia with 1.5 per cent, and respiratory failure with 2 per cent by volume in the alveolar air. Since chloroform is noninflammable it is frequently used, especially in England, when a noninflammable inhalation anesthetic is desired, and, because of its volatility, it is often used in the tropics or in warm climates. Not many physicians in the United States care to use chloroform to produce full surgical anesthesia. The use of chloroform in obstetrics has remained relatively frequent in this country, probably because the analgesic rather than the anesthetic effect is utilized, and the short period of induction makes it especially valuable. The premedication and precautionary measures used with chloroform are similar to those used with open drop ether. Chloroform is dropped on the mask as rapidly as the patient tolerates it, starting with 3 drops for the first minute and doubling the rate each succeeding minute for the first 4 or 5 minutes. As soon as possible castor oil should be instilled into each eye and a nasal catheter inserted into one nostril, supplying oxygen at approximately 1,000 cc. per minute; administration should continue at a rate necessary for the desired plane of anesthesia. During the induction and recovery periods, the patient should not be stimulated in any way, as this may lead to epinephrine intoxication and subsequently to ventricular fibrillation. Should bradycardia or irregularity in the pulse occur, the drug should be discontinued immediately. The danger of overdosage is ever present; therefore extreme caution should be used in administration.

Chloroform may be used for all types of surgery in which a potent anesthetic agent is required or as a preliminary induction agent for shortening the first and second stages of ether anesthesia. It has been widely used in obstetric analgesia and also for operations in which a noninflammable anesthetic is necessary. The induction period is rapid, and relaxation, particularly of the abdomen, is excellent. The greatest disadvantage to the use of chloroform is its extremely narrow margin of safety. Heart, liver, or kidney diseases, debilitation or emaciation, hypertension or hypotension are all contraindications to the use of chloroform. Oxygen should be supplied with chloroform if at all possible, and the use of epinephrine is contraindicated. A rather recent editorial[4] stated:

> It appears now that fear and distrust of this drug have been based upon incomplete knowledge and misconceptions out of all proportion to the facts. The group in anesthesia at the University of Wisconsin has presented a thorough study on chloroform such as is usually made in introduction of a new drug. . . . After a long experience of pleasant, convenient, rapid and effective induction, the merits of this nonexplosive drug should not be clouded by unsubstantiated prejudice. Investigation should be continued in other clinics, especially in an endeavor to find one or more other drugs which may be used with chloroform to counteract any of its harmful reactions.

ETHYL CHLORIDE

Ethyl chloride is a highly volatile colorless liquid with a chloroform-like odor. It boils at approximately 54 F. The drug is a halogenated hydrocarbon; it is easily inhaled and produces unconsciousness, analgesia, and surgical anesthesia very rapidly. Because of its volatility ethyl chloride should be kept in sealed glass or metal tubes in a cool place remote from fire and protected from light. The open drop method of administration is considered to be the simplest and safest, particularly for the beginner. The closed method permits the use of oxygen, but the depth of anesthesia is difficult to maintatin. Analgesia occurs when the alveolar air concentration reaches 2 to 3 per cent, and anesthesia with a concentration of 5 to 6 per cent. The recommended drugs for premedication are morphine and atropine or morphine and scopolamine. The technic of open drop administration of ethyl chloride, the precautionary measures, and the preparation of the patient are similar to those used in ether administration. It is advisable to hold the ethyl chloride ampule several inches above the mask and to drop the liquid rather than to spray it. The initial rate is 1 drop every 3 to 4 seconds with a gradual increase to 1 drop every 2 or 3 seconds until hyperpnea develops. If open drop ether is to follow, it should be added as rapidly as the patient can tolerate it. Ethyl chloride possesses a narrow margin of safety, and it may cause renal or hepatic damage if given over a prolonged period. It depresses the circulatory system and may cause cardiac failure; thus circulation may fail before respiration. Circulatory disturbances or disease, procedures requiring more than several minutes, the presence of acute respiratory infections, and the use of the cautery are all contraindications to the use of this drug.

Ethyl chloride is used as an induction agent to shorten the first and second stages of ether administered by the open drop route and as an analgesic and anesthetic agent for minor surgical procedures of short duration. Adriani's[5] comment on the use of

ethyl chloride is "Avoid use of this drug for inhalation anesthesia unless absolutely necessary." Anoxia or respiratory obstruction should not be tolerated under any circumstances, and epinephrine should not be administered in conjunction with ethyl chloride since both drugs increase the irritability of cardiac tissue. In a series of 2,500 cases Deming[6] used ethyl chloride in preference to Vinethene for induction. Ethyl chloride induction caused less increase of upper respiratory secretions, no liver damage was observed, and circulatory collapse did not occur.

SUMMARY

A comparison of the advantages and disadvantages and technics of administration of Vinethene, chloroform, and ethyl chloride has been made. All three agents are found to be wanting in one or more respects. There is need for further investigation of these anesthetics, and attempts should be made to overcome undesirable effects by the addition or discovery of a newer, safer drug.

1020 Seaboard Bldg.

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Cyclopropane and Helium

ROBERT L. THOMAS, D.O.
Columbus, Ohio

Principal consideration in this discussion will be given to the practical use of cyclopropane and helium as anesthetics. Complete information upon their history, pharmacology, chemistry, et cetera is readily obtainable in the literature on the subject.

CYCLOPROPANE

Cyclopropane is one of the most potent and useful gaseous inhalation agents which has stood the test of extensive clinical trial. Statistics on experience of over 20 years' use of this agent have been built up, and it now enjoys wide acceptance. Its desirable and undesirable characteristics have been probed, and cyclopropane has found its rightful place in the armamentarium of the anesthesiologist.

Among its advantages are that it is a rapid acting, pleasant, and nonirritating gas in anesthetic concentrations. It has a rapid, pleasant induction because of its potency and freedom from irritation to the respiratory passages. The anesthesia may be quickly lightened or deepened during the maintenance phase, and the concentration required for anesthesia allows the use of high partial pressures of oxygen. The elimination of nitrogen is unnecessary, so that the closed system may be used from the outset. Recovery is rapid and most of the drug is eliminated in about 10 minutes. It does not enhance acidosis, elevate blood sugar, or decrease renal or hepatic function. Finally, it decreases tidal volume and produces quiet respiration, and it is easy to induce apnea for facilitation of controlled respiration, when indicated.

Among its disadvantages may be listed its relative expensiveness, particularly if a completely closed system is not employed. It increases cardiac irritability and causes arrhythmias, particularly in the deeper planes, along with depression of respiration and accumulation of carbon dioxide in the blood stream, the latter often leading to an elevation of blood pressure and increased capillary oozing. Close observation is necessary, or the patient may pass easily from stage three to stage four. It is inflammable, and anesthetic concentrations are explosive when mixed with air or oxygen. Muscle relaxation is secured with difficulty in some cases; and to attain satisfactory relaxation, supplemental agents usually must be used. Nausea and vomiting are not unusual, and excitement or emergence delirium may occur during the recovery period. Many authorities recommend decreasing the amount of premedication if cyclopropane, with or without supplemental agents, is to be used, because overpremedication enhances the respiratory depression frequently observed with this drug. Facilities for endotracheal intubation should always be at hand when using cyclopropane, as laryngeal spasm during induction and recovery is relatively common and may require intubation.

In extensive clinical trial, cyclopropane has found its greatest field of usefulness in the shorter operative procedures not requiring profound relaxation and in thoracic surgery where quiet respiratory excursions and high oxygenation are important. Cyclopropane is a very useful agent for supplementation in regional methods of analgesia. Since the advent of muscle-relaxant drugs in clinical anesthesia, the shortcomings of cyclopropane with regard to satisfactory muscular relaxation have been overcome. Cyclopropane is one of the agents which carries a lesser hazard for patients in shock from trauma or hemorrhage. It is also clincially useful for patients who have severe metabolic disturbances such as diabetes, nephritis, and hepatitis.

I shall consider briefly a few points which I have found to be of use in avoiding some of the dangers and pitfalls which may occur with the use of cyclopropane. There are certain general principles which should be adhered to in using any anesthetic agent, but these principles are, perhaps, more important with cyclopropane than with most other agents. First is the maintenance of an unobstructed airway, whether by positioning the head to keep the tongue forward, by pharyngeal

airway, by the removal of secretions, by suction, or by the use of an endotracheal tube. I have mentioned the frequency of laryngeal spasm with the use of cyclopropane, and I feel that when using it, it is good insurance to intubate patients at the least provocation; at least a laryngoscope and endotracheal tubes should be at hand for immediate use when indicated. Second, adequate respiratory exchange should always be maintained. Adequate pulmonary ventilation is important because sufficient oxygen must reach the alveoli. Since cyclopropane is used with high oxygen concentrations this is not difficult, but since cyclopropane tends to cause respiratory depression, the importance of sufficient oxygen reaching the alveoli must not be overlooked. Equally as important as adequate alveolar oxygenation is the concomitant elimination of surplus carbon dioxide, which can be assured only by adequate pulmonary ventilation. With a patient anesthetized by such a depressing drug as cyclopropane, the normal respiratory response may be lacking owing to depression of reflexes, and the only way to eliminate carbon dioxide will then be by manually controlled respiration. Strict attention to making the patient breathe is imperative.

It is equally important to maintain an even level of anesthesia. It is well to predetermine the plane of anesthesia in which the patient should be maintained and endeavor to reach this level rapidly and evenly and then maintain it, unless the progress of the operation demands a change in the depth of anesthesia. Anesthesia unevenly maintained promotes the activation of reflexes such as vomiting, swallowing, secretion of mucus, straining and its resulting obstructive spasm, and other dangerous effects. This is especially important when cyclopropane is used in obstetrics, when the patients are often not adequately premedicated and have residual stomach contents. Respiratory paralysis with cyclopropane is insidious and may prove fatal. The technic of allowing a continuous flow of a small amount of gas into a closed circuit may eventually build up a dangerous concentration. The patient should be closely watched because there is a wide variation in the amount of cyclopropane required for anesthesia. It is easy to forget that the flow meter is turned on. If deeper anesthesia is required it is safer to turn a rapid flow of cyclopropane into the mixture for a measured period rather than to leave a supposedly harmless slight flow running in indefinitely.

Cyclopropane shock, an ill-defined clinical entity, is thought to be the result of the effects of retention of excess carbon dioxide in the blood stream. It can occur with any agent when there is depression of respiratory exchange without controlled ventilation. These patients go into circulatory collapse postoperatively, which can be very alarming, and it may be difficult to differentiate this shock from that which is caused by the surgical procedure, especially major abdominal or thoracic operations which are accompanied by trauma and blood loss. Most of these cases are probably from mixed causes, which emphasizes the advisability of continuous pulmonary ventilation throughout the anesthetic. Since this is the only presently reliable way of removing excess carbon dioxide, it is obvious that the use of efficiently acting soda lime is of paramount importance.

Ordinarily, with cyclopropane there is no particular cause to be alarmed about mild cardiac irregularities; these are observed frequently and apparently do not produce any damage or ill effect. However, the sudden onset of severe ventricular tachycardia, with a sudden rise of pulse rate to 120 or more, is an indication that the heart is sensitive to cyclopropane and that further attempts to administer it may lead to ventricular fibrillation. This complication seldom occurs, but when it does develop, cyclopropane should be discontinued in favor of another agent, preferably by addition of ether vapor or ethylene. The judicious use of intravenous procaine has been found helpful in eliminating arrhythmias, especially those occurring during intrathoracic procedures. Owing to the sensitized cardiac conduction mechanism which accompanies cyclopropane administration, the use of epinephrine, ephedrine, and similar drugs should be avoided.

The explosive nature of cyclopropane-oxygen mixtures requires that the anesthetist be ever alert to conditions which might remotely be the cause of igniting such a mixture. To have an explosion, a source of ignition must be brought into contact with an explosive mixture. In the operating room, fortunately, these conditions do not often occur concurrently, and explosions are the least common of the complications of anesthesia, yet they are the most dramatic and the most notorious. The hazardous zone is never more than a few inches away from the mask or the gas machine. Static electricity is the most difficult source of ignition to control and has been the cause of the great majority of anesthetic explosions. Every anesthetist should have a working knowledge of static electricity and its causes and become acquainted with existing information about its production and its control. Conduction materials, intercouplers, controlled humidity, and the like should be studied and used where indicated. With this knowledge and its application, the explosion hazard can be controlled.

HELIUM

Helium, a very light (atomic weight 4), inert gas, has limited usefulness in anesthesia and in the treatment of some cardiac and pulmonary diseases such as asthma. Becauses of its low density, very little effort is needed to move it in the tracheobronchial tree.

Helium is said to be helpful in some situations where laryngeal spasm or mechanical obstruction to the airway (such as in thyroid enlargement) occurs during inhalation anesthesia. The anesthetic atmosphere is diluted with helium, which presumably acts as a vehicle for the transport of the gases through narrow passageways. I have found this procedure helpful on rare occasions.

Helium has been used experimentally as a diluent for cyclopropane-oxygen mixtures in an effort to minimize or eliminate the explosibility of such mixtures. Its successful application in clinical anesthesia has been very limited because of the ever-changing demands for variation in potency of such mixtures and because of lack of a dependable indicator for determining whether or not the mixture in use at a given moment is within the explosible range. Although helium is on the anesthetic gas apparatus I rarely find use for it for the above reasons.

SUMMARY

Cyclopropane is a valuable agent for the anesthesiologist who has a working knowledge of its desirable properties and its possible dangerous effects, who respects its potency, and employs it with sound judgment. Its use in inhalation anesthesia in poor risk cases, either as an induction agent or for maintenance

alone or in combination with other agents, has found wide acceptance. Adequate pulmonary ventilation and alertness for arrhythmias and tachycardia are mandatory. Helium has found a limited field of usefulness for the anesthesiologist.

1527 Northwest Blvd.

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Nitrous Oxide and Ether

MORRIS L. STEIN, D.O.
Philadelphia

Nitrous oxide and ether are two of the oldest anesthetics, having been used successfully over 100 years ago. The fact that these agents have been in continuous use for a long period implies that their advantages far outweigh their disadvantages.

NITROUS OXIDE

Nitrous oxide is a colorless, inert gas having a sweet nut taste and odor.[1] Since its specific gravity is 1.53, it is heavier than air and, in addition to the usual methods of administration, can be given by gravity flow. It will not explode, nor is it flammable; however, it will support combustion, even in the absence of oxygen. The modus operandi of nitrous oxide is through its extreme solubility in the blood stream. It is merely a simple solution of a gas in a liquid (nitrous oxide in plasma) which displaces nitrogen, since nitrous oxide is 15 times more soluble than the nitrogen. There is no chemical combination as there is with oxygen and hemoglobin. The gas passes rapidly through the alveolar membrane, 85 to 90 per cent being eliminated by the same route, the gas being unaltered in any way by the body. The remaining 10 to 15 per cent passes through the skin. Elimination by way of the lungs requires only 2 to 3 minutes and in that time the patient has fully awakened.

Pharmacologically, nitrous oxide produces very little effect on the physiology in the presence of adequate oxygen.[1] However, these alterations should be mentioned: Metabolism is slightly depressed, the temperature regulating mechanism is depressed, and the pharyngeal reflex is slightly depressed. In over 6 million nitrous oxide anesthesias only one death was reported due to this agent.[2]

From the patient's viewpoint, nitrous oxide is the most pleasant induction agent available. It is nonirritating, has a pleasant odor, and induces anesthesia rapidly, with a minimum of excitation or other disagreeable symptoms.

Many physicians argue that nitrous oxide is not an anesthetic but an asphyxiant. This can be disproved by the simple experiment of administering to a patient a mixture of 20 per cent oxygen and 80 per cent nitrogen. No anesthesia results from this mixture. By substituting 80 per cent nitrous oxide for the atmospheric nitrogen, most patients will enter the analgesic state and some will become profoundly anesthetized, proving that nitrous oxide is a true, although weak, anesthetic.

Nitrous oxide is said to have a potency of about 15 per cent; that is, with adequate oxygen it is capable of carrying the patient just below the middle of the first plane of the third stage. Without adequate oxygenation, serious neurologic sequelae may result; but it would be unfair to incriminate the agent without first ascertaining whether the damage has resulted from a prolonged hypotension causing a stagnant anoxia, hemorrhage causing anemic anoxia, or from too deep an anesthesia resulting in altered cellular oxidation known as histotoxic anoxia.

Many patients can be maintained satisfactorily with concentrations of 30 to 40 per cent or even 50 per cent oxygen; 15 per cent of all patients will fall in this class.[3] On the other hand, the Army Air Forces showed that combat fliers at 10,000 feet suffer no bad effects from the reduced oxygen tension of only 14.25 volumes per cent and that at 11,000 and 12,000 feet, normal individuals tolerate as low at 12 to 13 volumes per cent.[4] The need of oxygen for these airmen must certainly be as great as, if not greater than, that required by the anesthetized, premedicated patient. Asphyxia does not occur with nitrous oxide anesthesia, as asphyxiation, by definition, implies not only restriction of oxygen availability but also obstruction to the normal elimination of carbon dioxide by way of the lungs.

Cyanosis can result only if there are 5 grams or more of reduced hemoglobin per 100 cc. of blood.[5] The anemic patient, therefore, will exhibit cyanosis much later and at a much lower plane of anesthesia than the plethoric patient. With less than 50 per cent hemoglobin content of the blood, cyanosis is rarely seen with nitrous oxide-oxygen anesthesia, and with 30 per cent or less, cyanosis is impossible to produce before death occurs. Clement[6] states, "The presence of cyanosis in nitrous oxide-oxygen anesthesia is not necessarily any indication of danger, nor is its absence any sign of safety."

The state of respiration is the most important single sign during nitrous oxide-oxygen anesthesia, and as long as an efficient type of respiration is evident, the patient can be in no danger. By efficient is meant continuous, regular, automatic respiration, with inspiration and expiration about equal, a little more rapid than normal, and with adequate volume exchange.

The signs of asphyxia during nitrous oxide-oxygen anesthesia have been well described by Allen and his associates.[7] They divide the asphyxial stage into three levels. The precrisis stage is characterized by an increase in rate and depth of respiration, a rapid pulse, and an increase in blood pressure. The asphyxial gasp-

ing stage is characterized by falling blood pressure, marked bradycardia—slow, full, bounding pulse—followed at the end of this stage by an increasing pulse rate. Breathing ceases, and after a short period of apnea, asphyxial gasping ensues. During those two stages the effects of hypoxia are reversible. The terminal stage is consummated rapidly and characterized by cessation of asphyxial gasping, a rapid drop in arterial pressure to zero, and a terminal slowing of the pulse with ultimate cessation.

These signs of asphyxia are masked by the various premedicant drugs used, atropine being the most dangerous since with its use there may be no development of a slow bounding pulse, although the terminal stage is reached. Scopolamine, a weak vagal depressor, allows the pulse changes to occur. Morphine does not affect pulse changes but depresses the blood pressure somewhat and alters its response to asphyxia; however, this effect is negligible if morphine has been administered 1 hour or more before the onset of anesthesia. The combination of morphine and scopolamine has fewer masking effects upon the signs of asphyxia than does the combination of morphine and atropine.

During nitrous oxide-oxygen anesthesia the greatest single hazard is the failure, once anesthesia has been established, to effect a gradual increase in the oxygen percentage of the mixture employed.[5] (Of course, this applies to all prolonged anesthesias, but particularly to nitrous oxide-oxygen anesthesia and to the subnormal risk). During induction, overdosage with nitrous oxide to the state of deep narcosis is manifested by such muscular spasticity that the need for oxygen is apparent; however, in the weak, debilitated, or anemic patient such spasm may not occur and just the opposite, flaccidity, may supervene.

Most failures of nitrous oxide-oxygen anesthesia result from too much oxygen being administered during induction and the early stages, and too little during maintenance. Once the desired level of anesthesia has been reached it must be remembered that the oxygen concentration must be increased as rapidly as possible without allowing the level to become sufficiently light that reflex movements occur. For example, if increasing the oxygen percentage from 10 per cent to 12 per cent allows reflex movements to occur, then a depressing level of anesthesia did not exist at the former concentration of oxygen. Later, during the same procedure, an increase to 15 per cent may not evoke signs of lightening anesthesia, and any concentration below 15 per cent would be unjustifiable. Failure to increase the oxygen concentration may lead to severe degrees of anoxia.

Crawley, Faulconer, and Lundy[8] point out:

> . . . great danger lies in the administration of mixtures of nitrous oxide and oxygen at low rates of flow, even though the original mixtures may contain an amount of oxygen that is well in excess of minimal requirements. . . . it has been consistently found in this study that for mixtures which contain more than 20 per cent oxygen, the oxygen made available to the patient will reasonably approach the original percentage of oxygen if total rates of flow are kept in excess of 6 liters per minute. The maintenance of high total rates of flow becomes more important as the percentage of oxygen in the original gas mixture becomes lower.

The oxygen apnea test is a simple test for determining whether the level of anesthesia is being maintained at a depressing level.[5] This respiratory test is based upon the fact that whenever a patient in need of oxygen is given an abrupt and profuse supply of it, temporary cessation of breathing, known as oxygen apnea, occurs. The duration of this apneic period is in direct proportion to the degree of oxygen deficiency. The chemical influence on respiration is by way of the chemoreceptors of the aortic and carotid bodies. Reduced oxygen tension during anesthesia results in reflex stimulation, by way of the vagus, of the chemoreceptor activity of the carotid and aortic bodies. An increase in the carbon dioxide content of the blood causes *direct* stimulation of the respiratory center in the brain. Administration of one or two breaths of 100 per cent oxygen removes this hypoxic stimulus, and a definite slowing of respiration occurs in about 7 seconds; it is indication that the patient has been suboxygenated and is in need of a greater oxygen concentration. If there is no effect on the respiratory rate or an increase in rate, it is evidence of lightening of anesthesia.

The semiclosed, fractional rebreathing method under positive pressure is by far the most efficient method of administering nitrous oxide-oxygen anesthesia. This method not only provides a greater saturation of the blood with oxygen but also advantageously employs carbon dioxide. That carbon dioxide is beneficial can be demonstrated by the fact that an 0.2 per cent increase of carbon dioxide in the alveolar air will double pulmonary ventilation, and a decrease of 0.2 per cent causes apnea.[9] Gellhorn[10] describes carbon dioxide as a "potent antagonist of anoxia." Further, according to Gibbs and his associates:[11]

> . . . normal brain function can be maintained in spite of low percentages of oxygen in the inspired air (as low as 2 per cent), provided the carbon dioxide tension of the body is maintained. . . . The addition of carbon dioxide to low oxygen mixtures permits maximal utilization of the available oxygen because of (a) increased pulmonary ventilation . . . (b) peripheral vasoconstriction and cerebral vasodilation . . . and (c) shift in the hemoglobin dissociation curve so that the blood unloads a greater proportion of its oxygen in the tissue. . . . The maintenance of the carbon dioxide tension of the brain is of equal importance for normal brain function.

The dangers of nitrous oxide-oxygen anesthesia are not from the action of the gas itself but from its maladministration. Success of administration is determined by the degree of proficiency of the administrator. In all anesthesias, the most essential single factor is the ability of the anesthesiologist.

ETHER

Ether, or ethyl ether, is the most widely used general anesthetic today and is the standard with which all newer anesthetic agents are compared. It has a potency of 100 per cent, inasmuch as it will carry the patient through all planes of surgical anesthesia to respiratory arrest without the necessity for preanesthetic medication or the intervention of oxygen deprivation. Ether, and also chloroform, will produce better muscular relaxation than any other inhalation agent.

Because of its great popularity, and because of the older misconceptions regarding the actions of ether, I think it is wise to consider the essential pharmacology of this agent and to review recent literature pertaining to it.

Central Nervous System.—

Initially, ether causes stimulation of the cerebral cortex which is probably responsible for the occurrence of marked delirium during induction, unless the induction is managed carefully.

Following this, cellular metabolism is decreased, the more highly developed areas of the brain being affected first. The cerebral blood flow is increased because of dilatation of the cerebral vessels. According to Adriani[2] the increase in intracranial pressure is not of clinical significance if the endotracheal route is used during maintenance with no obstruction to respiration. The medullary centers are lastly affected and only after a much larger dose has been given than is necessary for surgical anesthesia. For this reason, ether enjoys a reputation of having a wide margin of safety. Respiratory failure precedes cardiac failure, and attempts at resuscitation after respiratory arrest present less difficulty than would be the case with many other drugs and agents which are toxic to the myocardium.

Cardiovascular System.—The heart is first stimulated, and the rate and output are increased owing to the increased epinephrine secretion. During maintenance the pulse is normal. Ether protects against the occurrence of epinephrine-induced arrhythmias during cyclopropane. The effect upon the myocardium had always been assumed to be negligible; however, Fisher, Bennett, and Allahwala[12] have demonstrated that in dogs, at least, "Ether causes marked dilatation even in subanesthetic levels. Beginning with blood levels corresponding to second plane anesthesia this dilatation exceeds the physiologic . . ." A greater degree of dilatation is produced than that caused by cyclopropane, but less than that caused by chloroform. Its use in cardiac surgery, for which it heretofore has been considered a safe agent, may have to be re-evaluated.

It is a known fact that endotracheal manipulations during general anesthesia cause electrocardiographic changes. Burstein, Lopinto, and Newman,[13] in a series of 109 cases, showed an incidence of 68 per cent. However, Converse, Landmesser, and Harmel[14] report a lowered incidence which they believe is due to their custom of ventilating the patient's lungs with 100 per cent oxygen before any endotracheal manipulations are performed, and they state that ". . . in the absence of anoxia, reflex cardiac disturbances during endotracheal manipulations are likely to be of infrequent occurrence and of a transient nature. . . ." However, in the comparison of agents, they found that four out of four patients in whom ether was the primary agent, classifiable electrocardiographic changes occurred, whereas only half of the twelve patients showed changes when cyclopropane was the anesthetic.

Blood pressure is usually unchanged except in deep anesthesia. Hershey, Zurifach, and Rovenstine[15] point out that the functional efficiency of the peripheral circulation can be interfered with by different anesthetic agents. During deep anesthesia when the effects of circulatory stress were superimposed, the changes were most pronounced with ether, least with cyclopropane, and intermediate with Pentothal. These changes took place essentially in the capillary bed and consisted of depression of the response of the precapillary sphincters to epinephrine, depression of vasomotion, sluggishness of circulation through the capillary bed, slowing of blood flow in the collection venules, marked arteriolar dilatation, and a drop in blood pressure. From this it appears that in the presence of hemorrhage or trauma, ether should not be used below the very lightest levels of anesthesia, and cyclopropane would be the anesthetic of choice. The peripheral vessels of the skin are dilated with ether, which causes the pink appearance of the skin and the increased capillary oozing.

Blood Constituents.—Because of its sympathetic effect, ether causes a constriction of the spleen, thus throwing into the general circulation an increased number of red blood cells.[2] Thus ether is advantageous in splenectomy. The total blood volume and plasma volume is reduced 10 to 15 per cent, and the number of leukocytes is increased, the maximum being reached in 48 hours. The hemoconcentration produces increased viscosity of the blood. The carbon dioxide combining power is decreased soon after induction and results in acidosis.

The blood sugar rises 100 to 200 per cent, with the maximum rise occurring during the first 15 minutes of anesthesia.[2] The rise is only slight thereafter. The mechanism of this blood sugar rise has been shown by Johnson[16] to be a central stimulation which is transmitted to the adrenal glands by way of the sympathetics, and these glands, by hormonal action, cause glycogenolysis in the liver. Following bilateral extirpation of the adrenal glands, hyperglycema does not occur. Nor does it occur with high spinal anesthesia. Thus it may be in order to recommend high spinal anesthesia in cases in which it is particularly desirable to avoid hyperglycemia. The hyperglycemia of ether anesthesia can be statistically reduced by the induction of ether anesthesia with thiopental sodium. Although earlier experiments by Watts[17] showed that morphine had no effect on ether hyperglycemia, and whereas pentobarbital reduced the blood sugar rise, later experiments[18] showed that in man morphine alone and morphine plus barbiturates as premedication for ether anesthesia did not influence the blood sugar level. Only thiopental induction prior to ether reduced the hyperglycemia.

Liver and Kidneys.—

Ether depresses liver function; however, the liver returns to normal in 24 hours.[2] Armstrong[19] showed that ether causes a greater degree of liver damage than trichlorethylene. There is decreased secretion of bile and bile salts. Liver glycogen is depleted almost 50 per cent after an hour of ether anesthesia after which the decrease is gradual.[2] These effects on the liver are intensified by oxygen lack and a poor nutritional status of the patient.

Ether suppresses urinary output, and stage three anesthesia is accompanied by oliguria.[2] Possibly this is due to pituitary release of antidiuretic hormone. During recovery, there is a compensatory polyuria, and in about 50 per cent of cases albuminuria is present, which may persist for a few days. No histologic changes are produced, unless some type of renal insufficiency is present prior to anesthesia.

During administration of ether, it has been found[20-22] that higher inspiratory concentrations of ether are necessary than is generally believed. The average concentration during surgery in the peritoneal cavity ranged from 6.1 to 13.7 volumes per cent. These investigators also have shown (by electroencephalographic studies) that lower concentrations of ether are necessary to produce the same level of anesthesia when nitrous oxide is present in the mixture. In the open drop method of anesthesia, they further demonstrated that the mere application of a mask to the face of a patient reduces the partial pressure of oxygen under the mask from that of the air in the room (153 mm.) to an average of 123 mm. The addition of ether to the mask further reduced the oxygen pressure to an average of 105 mm. It was found that during this type of anesthesia there were frequent sig-

nificant reductions in arterial oxygen saturation, and that these reductions were directly related to the changes in oxygen tension that existed under the mask. Therefore, in this method of ether administration, perhaps the most frequent in ether anesthesia, as little as 500 cc. per minute of oxygen should be added to the mixture under the mask, thereby restoring oxygen saturation of the arterial blood to normal.

CONCLUSIONS

In this review of the literature, I have attempted to point out some of the more important advances in the use of the agents, nitrous oxide and ether. These advances have changed some concepts, augmented others, and some concepts have merely been explained. All this work can only lead to a better understanding of the principles involved, which in turn makes surgery safer for the patient. As Laycock[23] states:

> . . . it is reflexes in the operative field that demand our attention, not merely the respiratory reflexes, or the swallowing, eyelid or eyelash reflexes, and certainly not the cough reflex. There are many different reflexes, some of importance in orthopaedic surgery, others in gynaecology, others in abdominal, and others in thoracic surgery. They are the guiding principles of all we do from the moment we start an anaesthetic until the patient wakes up. We must understand them, look out for them, nurse them, leave them alone, depress or abolish them. In fact, in using anaesthetic agents we might even say we need to know only three things about them—their effect on consciousness, their toxicity, and their actions on the reflexes.

6437 Ridge Ave.

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Biochemistry in Anesthesia

GRACE B. BELL, D.O., M.S.
Department of Biochemistry and Nutrition
College of Osteopathic Physicians and Surgeons
Los Angeles

The basic sciences in the past dealt with rather sharply delineated fields of study. The borders between such fields of study as chemistry and anatomy were often jealously guarded by the teachers of these subjects, lest perhaps their territories be encroached upon. Today we find the barriers are disappearing, that the interdependence of basic sciences upon one another and of the therapeutic fields upon the basic sciences are being strongly emphasized.

Biochemistry must often encroach on such subjects as physiology, pharmacology, and bacteriology. Biochemistry appears to relate itself to all fields of therapy. It would be difficult for a biochemist to train a medical student adequately in biochemistry without some discussion of the effects of anesthetic and other therapeutic agents upon the chemical processes of the body.

To say that the modifications of the chemical processes of the cell which result in narcosis are clearly understood would be indulging in wishful thinking. We have a certain degree of understanding which is based on noting the changes which occur when anesthetic agents are administered. These changes might be termed symptomatic. The manner in which these changes are produced is not always clear.

General anesthetic agents, including ether, chloroform, and barbituric acid derivatives, produce chemical changes which vary somewhat between the different anesthetic agents, but which for each agent are fairly constant when administered to the human subject. The changes which have so far been demonstrated include alterations of the carbohydrate balance in the body. Glycogen stores of liver and muscle are depleted; blood glucose is elevated. In spite of the hyperglycemia the oxidation of glucose is as a rule inhibited by anesthetics.

Carbohydrate metabolism is so fundamental in supplying the energy needs of the cells that any disturbance of it is apt to have widespread effects. The patient subjected to anesthesia is often in a state of poor glycogen reserve. This may be the result of a period of fasting or semifasting without adequate attention to nutritional needs. Anxiety, fear, and nervous tension contribute to lowered glycogen reserves, probably largely through increased production of epinephrine by the suprarenals. If there is excessive

muscular effort and excitement during induction the rate of glycogen breakdown is also accelerated.

Preinduction medication may influence the degree of hyperglycemia produced by an anesthetic agent. Bass and others[1] studied the influence of three different combinations of premedication and induction on ether hyperglycemia. They used morphine premedication followed by nitrous-oxide induction of ether anesthesia, premedication with both a barbiturate and morphine followed by nitrous oxide induction of ether anesthesia, and premedication with a barbiturate and morphine followed by Pentothal-nitrous oxide induction of ether anesthesia. In each series they found that the premedication had no significant effect on blood glucose levels. After 22 minutes of anesthesia with ether blood glucose levels showed a mean increase of 30 mg. per 100 cc. in the first series (morphine alone). The second series of premedication with morphine and barbiturate had a mean increase of 28 mg. per 100 cc. In the third series the barbiturate and morphine premedication followed by Pentothal-nitrous oxide induction showed a mean increase of only 12 mg. per 100 cc. of blood, a statistically significant inhibition of ether hyperglycemia.

The patient who has a normally functioning liver, and whose carbohydrate metabolism is essentially normal, is probably not affected adversely by the hyperglycemia of anesthesia. If there is hepatic damage with decreased glycogen storage the depletion of hepatic glycogen may result in liver damage by the anesthetic agent. In surgery under general anesthesia for biliary tract disease, severe and even fatal hypoglycemia may occur after surgery as a consequence of exhaustion of hepatic glycogen because of associated but not readily detectable hepatitis. The presurgical care of such patients might well include measures designed to enhance hepatic glycogen storage whenever this is possible.

The administration of most anesthetic agents influences the hydrogen ion concentration of the blood plasma. During induction with ether or chloroform the hydrogen ion concentration rises suddenly, then increases gradually during maintenance of anesthesia. In the latter stages of anesthesia, ketosis and ketonemia frequently occur. These probably do not contribute to the acidosis of early anesthesia.

Electrolyte concentrations inside the cells, in the extracellular spaces, and in the blood plasma are influenced by some anesthetic agents. Potassium ion which is normally largely confined to the intracellular material shifts to the extracellular fluid, as does magnesium ion. At the same time calcium ion shifts from extracellular to intracellular fluid.

Anoxia of the central nervous system has been shown to be present during anesthesia produced by barbiturates and other anesthetic agents. This was demonstrated by McClure and his coworkers.[2] Other workers have shown that anoxia is more prominent in the cerebral cortex than in the lower nervous centers. Together with decreased uptake of oxygen there is lowered utilization of dextrose.

Respiratory acidosis which is the result of inadequate removal of carbon dioxide in the lungs may occur in anesthesia. Carbon dioxide diffuses from cells into the blood where it influences the pH of the blood through the various compounds which it forms. In general, in the absence of metabolic deviations low blood carbon dioxide is accompanied by high blood pH, or alkalosis, while elevated blood carbon dioxide results in lowered blood pH or acidosis.

Accumulation of carbon dioxide not only affects the pH of the blood but has profound physiologic effects. The depressant effect of this metabolite is enhanced by ether anesthesia, and perhaps by other anesthetic agents as well. The changes in pH affect the fluid and electrolyte balances between the blood and tissues. Gabbard and associates[3] have emphasized that the accumulation of carbon dioxide in the blood may be an important causative of such anesthetic accidents as cardiac arrhythmias, precipitous drops in blood pressure, convulsions, and sudden cessation of heart and respiratory function. Johnstone[4] reported that elevated blood carbon dioxide during cyclopropane anesthesia in man produced ventricular arrhythmias which disappeared when the blood carbon dioxide level was lowered.

In anesthesia changes in pulmonary ventilation may result from the effects of the anesthetic agent on the respiratory control, as in the case of depression by cyclopropane or barbiturates, or the stimulation by ether. Respiratory studies have been made during anesthesia with various agents. Patrick and Faulconer[5] in a study of six cases found that ether anesthesia had little effect on the carbon dioxide tension of arterial blood, while in seven cases of Pentothal sodium and oxygen anesthesia the arterial blood carbon dioxide was elevated only when respiration was markedly depressed by deep narcosis.

Changes in pulmonary ventilation may result from surgical procedures employed during the period of anesthesia. When the surgical procedures involve extrathoracic structures spontaneous adequate pulmonary ventilation can usually be maintained. In surgery of thoracic structures carbon dioxide has been found to accumulate in the arterial blood and acidosis has developed in cases in which there was no assistance to respiration. The dynamics of ventilation are interfered with to some extent whenever the thorax is opened. During pneumonectomies and lobectomies there are several operative factors which interfere with normal ventilation. The opening of the pleural cavity changes the pressure relations which are normally in effect. The packing or other procedures used on the diseased lung while blood flow is maintained interfere with the gaseous exchange so that the composition of the blood is not changed as it passes through the pulmonary circulation of certain areas. To compensate for this the sound lung will have to be hyperventilated. In all anesthetic procedures it is well for the anesthetist to keep in mind constantly the maintenance of normal carbon dioxide tension in the lungs, together with normal oxygen saturation.

CONCLUSION

These effects should not be interpreted as being the chemical changes which induce narcosis, but rather as being produced by the influence of anesthetic agents on cellular metabolism, or by the physiologic effects of such metabolic changes as these may influence hormonal or nervous control in the body as a whole.

2434 Glendower Ave.

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Infant Resuscitation

MAHLON L. PONITZ, D.O., F.A.O.C.A.
Detroit

THE AGE OF FETAL SALVAGE

The tremendous decrease in fetal mortality during the first year of life, from 100 per thousand live births in 1915[1] to an estimated 28 per thousand live births in 1953,[2] suggests that the age of fetal salvage may have been reached. Of this number 4.6 were due to congenital malformations, 11.1 to prematurity, 3.6 to birth injuries, 1.6 to asphyxia and atelectasis, and the remainder were unclassified.[3]

There has been little change in the death rate associated with birth and the immediate neonatal period. The mortality rate during the first week of life is still 52 per cent of the mortality for the entire first year, and that during the first day of life remains 29 per cent of the total of the first year.[3] If these facts are interpreted to mean that the pediatrician has performed his duties more satisfactorily than have the obstetrician and the anesthesiologist, who are charged with the immediate care of the infant during labor, delivery, and the early neonatal period, then the responsibility of the obstetrician and anesthesiologist in the attainment of a greater fetal salvage must include not only safe delivery of the infant but also safe conduct through the perilous first few hours of life.

THE INITIATION OF RESPIRATION

Barcroft[4] concludes from his studies of sheep that respiration occurs when the sum total of stimulation from different sources is sufficient to overcome central inhibitions. Stimuli are chemical, from changed relationships between oxygen and carbon dioxide concentration at the respiratory center, and sensory, from afferent nerve impulses originating in the skin, mucous membranes, and muscles. These are augmented in an auxiliary manner by stimuli from the chemoreceptor mechanism of the carotid and aortic ganglia activated by relative anoxia. The responsiveness of the respiratory center is dependent upon adequate oxygen supply to the cells of the center.

Breathing of an immature type, that is, variants of Cheyne-Stokes breathing, occurs in a high percentage of normal newborn infants in an atmosphere of air alone. Regular rhythm can be brought about by increasing the oxygen concentration. The majority of premature infants breathe while in an atmosphere of air as if suffering from oxygen lack, yet do not have a strikingly reduced arterial oxygen saturation. However, administration of added oxygen corrects this type of breathing.

Most infants breathe spontaneously and immediately or with only slight delay and carry on adequately even though their respiratory rhythm is frequently irregular. The majority of those who experience some difficulty are relieved by oxygen administration. These are the infants whose respiratory centers have been depressed by a moderately severe degree of anoxia from various causes during birth.

RESPONSIBILITIES OF ANESTHESIOLOGISTS

There has been a revival of interest in the etiology and the clinical and pathologic manifestations of various types of pulmonary obstructive lesions. It is not the scope of this paper to make a study of this important phase, but one consideration that alters the entire concept of infant resuscitation is that of the hyaline membrane (vernix membrane, asphyxial membrane).[5] It is recognized that the pulmonary hyaline membrane is a major factor in deaths from respiratory causes in the newborn period. The membrane is plastered against the walls of the respiratory bronchioles, the alveolar ducts, and the alveoli. Simply stated, there is plugging of the ducts which produces secondary atelectasis. Blystad and his associates[6] suggest that the membrane is protein in nature and undoubtedlly comes from amniotic fluid. Air breathing of some duration is believed essential for its formation, since it is seldom found in stillborn infants.

Clinically, the picture is seldom varied. Breathing is quite normal for several hours after birth, and then evidence of dyspnea appears. The main visible sign is retraction of the sternum and the costal margins with some grunting and cyanosis. Death may occur rapidly, or the condition may exist for several days and then suddenly improve. In the newborn infant with respiratory difficulty associated with atelectasis there are factors favorable to airtrapping which may lead to emphysema. Atelectasis is rarely pure atelectasis. The obstruction is in many small bronchioles. Some are completely blocked; others become so. In bronchioles that are partially blocked air enters the alveoli but is hindered in its exit, thus giving the picture of atelectasis and emphysema simultaneously. Other pathologic changes are congestion, edema, hemorrhage, and tissue degeneration, all occurring as a result of anoxia.

During labor and delivery the prevention of fetal asphyxia is the problem of the anesthesiologist. The fetus must be assured sufficient oxygenation by administration of oxygen to the mother during labor. Depressing drugs should be used cautiously, or not at all. Gaseous inhalations of nitrous oxide, cyclopropane, and Trilene must contain an adequate amount of oxygen. When conduction analgesia is used, maternal blood pressure must be maintained above 80 mm. of mercury. When below this point for 5 minutes or longer, fetal anoxia is always evident. Certain systemic

diseases of the mother are also causes of anoxia and must be anticipated. The most common cause of fetal anoxia due to interference with the passage of oxygen from mother to fetus is probably premature separation of the placenta. It is important to individualize therapy in each case.

RESUSCITATION

Asphyxia is failure of a person to breathe after 30 seconds. Flagg[7] divided asphyxia into three stages. First stage or stage of depression: The infant's breathing is slow and irregular with tendency to cyanosis. Second stage or stage of spasticity: Respirations are irregular and gasping with cyanosis or pallor. Reflexes are sluggish. Third stage or stage of flaccidity: Respirations cannot be demonstrated, the jaw is relaxed, the glottic structures are collapsed and the laryngeal reflex is absent. Circulation is certainly failing.

There are four salient principles of resuscitation to be remembered when caring for the asphyxiated newborn infant: (1) The air passage must be cleaned. (2) The body temperature must be maintained. (3) The infant must be handled gently and as little as possible. (4) Oxygen must be given intermittently under pressure or continuously at atmospheric pressure.

At delivery, as soon as the head is born, the nasopharynx and nares should be suctioned. As soon as delivery is completed, the infant is placed in the inverted position, the trachea is milked, and the mouth, nasopharynx, and nares are again suctioned with a small soft catheter (French number 14). The cord is clamped and cut when it ceases to pulsate, the baby is placed in a heated bassinette in the Trendelenburg position, and oxygen is administered at the rate of 4 liters per minute.

Should the infant show signs of obstructive breathing, the pharynx is again suctioned and the tracheobronchial tree is aspirated. (Suction of the gastric contents is also very important). The use of an infant laryngoscope readily exposes the larynx, and a soft number 9 tracheal catheter is passed into the trachea and mild suction applied. At this point, intermittent oxygen is supplied intratracheally. It is well to discontinue the intermittent positive pressure every few minutes in order to observe signs of spontaneous respirations. The tracheobronchial tree is suctioned at intervals with oxygen continued. At the first sign of regular respiration, positive pressure oxygen is stopped, and continuous oxygen flow is substituted. When the child's color, respirations, and general appearance are satisfactory, extubation is accomplished with careful suction at the same time, and the child is placed in an incubator and a continuous flow of oxygen is supplied at the rate of 8 liters per minute. The child should be observed for the next 2 to 3 days and suction applied when necessary. Periodic inhalations of Carbogen (95 per cent oxygen and 5 per cent carbon dioxide) stimulate respirations and are often beneficial. Helium-oxygen mixtures are also sometimes indicated.

It is my opinion that infant bronchoscopy is beyond the scope of this paper since it is another of the very specialized technics based on postgraduate lectures and demonstrations in bronchoesophagology. Authorities on this subject agree that the Jackson laryngoscope and bronchoscope are the best endotracheal instruments available for resuscitation. Flagg,[8] who is one of the pioneers in the field of resuscitation, states, ". . . nothing is finer than the Jackson instrument."

It should be emphasized at this point that rough handling of any nature is to be condemned. This includes severe spanking, hot and cold tub baths, swinging, rectal dilation, compression of the chest, and the like.

Bloxsom's oxygen-air lock is an ingenious device, but there is not universal agreement that the physiologic premises on which it is based are sound. The Drinker respirator, as an initial method of respiration, is of no value. For infants who have breathed and who are depressed, the air-lock and respirator are of value. Absence of negative intrapleural pressure in the infant who has never breathed is also the reason the Shafer and Silvester methods of artificial respiration are of no value in asphyxia of the newborn. Acceptance of a machine as a replacement for personal responsibility in medical practice is dangerous, not only from the standpoint of the patient's welfare, but from the standpoint of anesthesiologic practice as well.

Certain drugs, including alpha-lobeline, Pituitrin, Coramine, picrotoxin, Metrazol, and caffeine have been employed in treating atelectasis or asphyxia of the newborn, but they are capable of producing more harm than good. These so-called stimulating drugs are not selective stimulators of the vasomotor and respiratory centers; rather, they stimulate those centers in the course of their action as generalized cerebral stimulators. The action of these drugs is unreliable; they often cause a short excitement stage or even convulsion, followed by a period of severe depression. These facts would appear to contraindicate the use of these drugs in the therapy of asphyxia neonatorum.

CONCLUSIONS

The administration of 100 per cent oxygen by mask or endotracheal tube is the treatment of choice in asphyxia neonatorum. The Jackson laryngoscope and bronchoscope are the instruments best suited for this work, although specialized training is necessary for their effective use. Flagg[7] repeatedly insists that if the glottic reflex is active, the infant will respond to oxygen. If the reflex spasm of the glottis is absent, the infant is in the third stage of asphyxia and dying. The primary pathologic change is complete atelectasis of the lungs. The associated anoxia produces a decrease in the concentration of oxygen, increased carbon dioxide and lactic acid tension, with resultant decrease in the hydrogen ion concentration of the fetal blood. Permanent damage may result from this type of anoxia. Extremely gentle stimulation externally may be applied in an attempt to stimulate the respiratory center with afferent impulses. Warmth should be maintained throughout; stimulatory drugs may do more harm than good.

SUMMARY

The present-day concept of asphyxia has been reviewed. A method of resuscitation has been given and several rules of caution advanced. Only by adherence to the previously mentioned contributions of investigators and authors and many more will we as anesthesiologists have a brighter outlook on "the valley of the shadow of birth."[9]

5435 Woodward Ave.

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Mechanisms of Local Anesthetic Action

ROBERT W. EARLE, B.A., M.S.
Executive, Department of Pharmacology
College of Osteopathic Physicians and Surgeons
Los Angeles

Studies of the mechanisms whereby drugs produce their effects have become increasingly important in recent years as the basic sciences have advanced. We are learning that fundamental discoveries in one field of study will have effects in all other fields and actually may, in due time, affect the manner in which physicians prevent or treat abnormal conditions.

There are several practical reasons why the mechanisms of drug action are important to the physician. While knowledge of all of the mechanisms may not be of immediate value or may not entirely apply to the local anesthetics, the main reasons are listed here for the sake of completeness.

The mechanisms of action may:

1. Serve as a starting point for the development of new drugs. For example, the development of BAL (British Anti-Lewisite) was an outgrowth of an understanding of arsenical poisoning.

2. Permit selection of alternate drugs in situations where the drug of choice is unsafe due to allergy, resistance, etc.

3. Allow selection of a more effective antidote. For example, the intestinal cramps produced by muscarine are better antidoted by atropine than by morphine because atropine works directly upon the nervous structures involved to relieve spasm rather than through a central action far removed from the site.

4. Permit selection of a drug having a greater degree of effectiveness against disease-producing organisms.

Any theory must be founded upon and tested against experimental observations. Unfortunately, the observations of the effects of local anesthetics on nerve cells are difficult to obtain and are, therefore, meager in amount. However, a fair body of information has been accumulated by study of the squid nerve axon, frog sciatic, and the metabolic pathways of brain tissue.

The basic observations are as follows:

1. Local anesthetics block transmission of the nerve impulse at relatively low concentrations.[1,2,3]

2. The affected nerves are blocked in a sequence based upon size and myelination. The smallest fibers, fortunately carrying impulses originating in pain receptors, are the first affected, followed by the next largest, transmitting temperature and touch, with the largest or motor nerves being the last affected. Myelination delays or decreases the onset of block.[4]

3. The nerve membrane potential is maintained at its original level while the nerve is blocked.[2]

4. The oxygen consumption of nervous tissue is reduced by local anesthetics, but at concentrations 10 to 100 times those required to produce block.[5]

5. Certain intracellular enzymes are inhibited, but again at concentrations ten to 100 times those required to produce block.

The theories that have been evolved to explain local anesthetic action must take into account not only the above observations but also what is known about the nature of nerves and of the nerve impulse. In the first place the nerve impulse is dependent upon the ability of the nerve cell to maintain an electrical potential on the membrane. When the nerve conducts there is a rapid, progressive discharge of this potential which then requires an appreciable interval to regain its original level. The nerve membrane has to be permeable to the nutrient materials required by the cell yet impermeable enough to prevent the spontaneous discharge of the potential. The potential is probably dependent upon the balance between sodium and potassium ions inside and outside the cell membrane. In fact, calculations on the potential obtained by differences in the sodium and potassium ions agree very closely with the potential experimentally obtained.[6]

The discharge of the membrane potential is accomplished by a structural alteration of the membrane itself which permits the inward diffusion of sodium ions and the outward diffusion of potassium ions. The cell probably maintains some mechanism (the sodium pump[6]) very near the membrane for the exchange of the sodium ions which have entered for the potassium ions which have left. While the excretion of sodium ions is being accomplished there is a gradual increase in the membrane potential to its original level and it is during this period that the cell's energy requirement is at its highest.[7] Since the local anesthetics produce block in lower concentrations than are required to affect the cellular enzymes, it is probable that the local anesthetic is reacting with the cell surface.

Reaction of the local anesthetic with the cell surface probably results in a decrease of the permeability of the membrane[8] to prevent the entrance of sodium and the potential is stabilized. A stabilization of the potential would reduce the necessity for excretion of sodium by the cell and would thereby reduce the energy requirement with a consequent reduction in oxygen consumption. A sidelight on this phase of local anesthetic action is the observation that these drugs increase the ability of nerves to resist the depolarization caused by anoxia.[9] The reason is simply that blocked nerves require less oxygen for their metabolic processes.

The factors involved in the alteration of the complex lipoprotein membrane by the local anesthetic to

render it less permeable are, as yet, even less defined than the effects upon nerve reaction.

The above theory, that local anesthetics act by decreasing permeability of the nerve membrane through an effect first upon the membrane and then indirectly upon the metabolic activity, may be examined in the reverse by postulating that metabolic activity is first influenced to then alter cellular permeability. Evidence in favor of this approach is based on the demonstrated effects local anesthetics have on oxygen consumption and on the cellular enzymes. As noted before, these effects are produced with concentrations ten to 100 times the concentrations to produce block.

Conditions could be postulated which would explain penetration of the drug on the basis that the lipoid nature of the nerve cell would accumulate the drug until the requisite level was attained. Such an approach would explain the observation that small fibers are blocked before large fibers because large fibers have more enzymes to be blocked and also a greater energy reserve. The objection to this approach is that the cellular enzymes are mostly involved in the recovery process so if the enzymes were blocked, the potential could not be re-established and the nerve would be depolarized while blocked, but actually, the reverse is experimentally observed.

A third theory[10] based on similarities of chemical structure between acetylcholine and most of the local anesthetics postulated that the transmission of the nerve impulse involved the reaction of acetylcholine with certain receptor groups and that this reaction was prevented by the local anesthetics. Acetylcholine is a substance known to be important as a neuroeffector substance at synapses[11] and by inference was thought to be important in transmission along the axon.

In more recent years this theory, although an attractive one, has been discarded because: (a) Acetylcholine has not been definitely shown to be important in axonal transmission, and (b) other compounds having similar structures are not local anesthetics.

In summary it may be said that the basic effect of the local anesthetics is to interfere with the transport of ions through the cell membrane. Whether this is accomplished by a primary effect upon the membrane or upon the cellular enzymes is still open to question. However the drugs of this group produce their effects, they are useful tools to elucidate the nature of peripheral nerves and of the nerve impulse. The understanding thus gained will provide a basis for attack upon a much more difficult problem and that is the nature of the central nervous system and the mechanisms whereby drugs produce their effects upon this organ.

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Diagnosis and Treatment with Continuous Conduction Analgesia

HELEN K. GAMS, D.O.
Houston, Tex.

INTRODUCTION

Conduction anesthesia, as the term is used in Pitkin's "Conduction Anesthesia,"[1] denotes interruption of nerve conduction distal to the site of application of a drug along the course of a nerve without disturbing the mental faculties of the patient or interfering with body metabolism. It includes local infiltration, nerve blocking, single injection and continuous spinal, caudal and peridural block, and refrigeration anesthesia as well as diagnostic and therapeutic nerve blocking. During the past decade increasing interest has been shown in the diagnostic and therapeutic use of conduction analgesia by the continuous routes. Basically the same anatomic and physiologic concepts, precautions, indications, and contraindications exist for the continuous technics as for the single dose or "one-shot" methods, and with a little practice the anesthesiologist becomes as adept in using the former methods as the latter. In all instances he should become routinized in his procedures so that his technics are smoothly performed, results consistently gratifying, and reactions conspicuous by their absence.

Numerous local anesthetic drugs are in use today. They all possess certain chemical, physical, and physiologic properties which necessitate judicious choice if they are to be used successfully. Most of them can be adapted for use in continuous conduction analgesia.

Thirty to 45 minutes before a continuous conduction analgesia procedure is started the patient should receive one of the barbiturates, such as Delvinal or Nembutal Sodium, to protect him from any reactions which he might exhibit because of individual sensitivity to the anesthetic agent and to prepare him psychologically for the block. At all times during the administration of the block one of the quick-acting barbiturates, such as Surital Sodium or Pentothal Sodium in a 2½ per cent solution, must be in readiness for immediate intravenous injection should a marked reaction to the anesthetic drug appear. The two most important resuscitative measures in the anesthesiologist's armamentarium—a means of immediate endotracheal intubation and a source of positive pressure oxygen—must ever be close at hand to combat the sudden, profound cardiorespiratory collapse which can,

unfortunately, complicate a rather simple diagnostic or therapeutic procedure.

TECHNIC

This paper will concern itself only with the anatomy of the region as related to the technical problems of conduction analgesia. Little need be said regarding the technic of spinal puncture. The patient is placed in the lateral recumbent position with knees flexed on abdomen and chin on chest, the skin prepared with tincture of Zephiran or Merthiolate, a sterile spinal drape placed over the patient, and the skin, subcutaneous tissues, and interspinal ligament infiltrated with 1 per cent procaine hydrochloride.

A 16 gauge sharp bevel Tuohy needle with a Huber point is inserted into the subarachnoid space at the desired level. The needle is inserted with the bevel pointing laterally in order to split, rather than cut, the dural fibers. When the needle is in the subarachnoid space, as felt by the dural "snap" or "pop," the curved bevel end is turned cephalad or caudalward depending on what level of analgesia is desired; the stylet is removed; and a No. 2 or 3 Bard ureteral, a polyethylene, or a vinylite catheter is inserted about 1 to 1½ inches beyond the tip of the needle. I find polyethylene catheters easier to handle and insert, and have less incidence of paresthesia upon insertion than when I use the stiffer vinylite variety. The tip of the needle is readily felt, offering slight resistance to the catheter as it passes through it. The catheter should be aspirated to be certain that it is well within the subarachnoid space. The Tuohy needle is then carefully withdrawn over the catheter with one hand while the catheter is gently threaded into the needle with the other hand. A 23, 24, or 25 gauge hypodermic needle is inserted into the distal end of the catheter connecting it to the anesthetic source by means of a one-way stopcock and syringe filled with anesthetic solution, or a two-way stopcock fastened to a 20 cc. Luer-Lok syringe and rubber tubing connected to a bottle of anesthetic solution. The 20 cc. syringe is used in an attempt to prevent contamination of the plunger of the syringe during repeated filling of the syringe.

Only 5 or 10 cc. of solution are aspirated into the syringe so that the lower end of the plunger is never outside of the syringe proper. To absolutely minimize the possibility of contamination, a fresh syringe can be used for each injection of anesthetic solution. A sterile gauze sponge is doubled up and placed beneath the catheter as it emerges from the opening in the skin to avoid sharp kinking. One strip of adhesive tape is placed across the sponge perpendicular to the long axis of the back and another longer piece follows the catheter up the back for a distance, bringing the catheter to a suitable place on the patient's abdomen or shoulder. If desired, between injections, the syringe can be removed and the distal end of the catheter closed with a sterile Luer-Lok needle cap. In order to avoid shearing the catheter within the subarachnoid space or shaving off particles of it, the catheter must never be retracted through the Tuohy needle.

Although the plastic catheters are guaranteed to be inert to tissue and tissue fluids, conservative users feel that they should be replaced every 36 to 48 hours for utmost protection of the patient and for assurance of patency of the proximal tip since granulation tissue tends to form about and occlude the tip if it remains within the body too long. Recent reports[2] mention leaving the catheter in place from 3 days to a week with no adverse effects. As a rule the catheter is easily and painlessly removed by gentle pulling; however, if any resistance to its removal is encountered, flexing the patient's back will widen the interspaces and facilitate its extraction.

The fractional spinal technic using a catheter is far superior to the method using a malleable needle for surgical anesthesia and is almost mandatory for use in conduction diagnosis and therapeutics. While the ordinary Bard ureteral catheter can be used, introduction of this catheter into the subdural space is often associated with one or more painful paresthesias, or the catheter is so stiff that insertion beyond the needle tip is very difficult or virtually impossible. Toxic tissue reactions result if a bit of the catheter is broken off in the patient.

Plastic catheters are made of a flexible, tough, yet pliable tubing which produces no tissue reaction, comes in a variety of sizes for introduction through smaller gauge spinal needles, and is relatively inexpensive. The plastic tubing is obtained from the manufacturer in 100 foot strands or in individual 36 inch lengths at a slightly higher price. These catheters are used but once and then discarded—a cost of approximately 15 to 30 cents per catheter, as compared to the initial cost of $4.50 for a ureteral catheter which can be resterilized and used several times (usually not more than six to eight) before it begins to crack or flake.

The question of sterilization of the plastic catheter has often been discussed and various means have been advocated. Most anesthesiologists are of the opinion that an agent to be used with safety within the subarachnoid space must be prepared for use by heat-pressure sterilization. However, polyethylene tubing will melt under this treatment and therefore must be sterilized by immersion in 1:1,000 aqueous Zephiran for 24 hours in each of two separate jars.

A vinylite catheter can be processed by autoclaving or boiling. When boiled and used immediately it remains fairly pliable; however, when autoclaved at 250 F. and 15 pounds pressure for 5 minutes, it becomes relatively stiff and springlike. In order to prevent kinking and facilitate removal of the sterile vinylite catheter from its sterile wrappings, one end of it (approximately 4 inches) should be placed in a test tube, the remainder curled in a fair-sized loop and then double-wrapped for autoclaving; or it can have a fine wire stylet inserted to insure patency and be curled into a Petri dish, taped shut, dated, and autoclaved. An assistant must open the Petri dish for the anesthesiologist who can then control the springlike action since the ends of the catheter can be seen.

It has been suggested that the wire stylet be left in place to prevent kinking in the epidural or subdural space. I can see only possible dangerous results from this practice. If the catheter cannot be inserted without a stylet in place, the patient is poorly positioned, or the anesthesiologist lacks the experience and skill needed for nontraumatic catheter insertion. If great difficulty arises, it is best for him to remove the needle and catheter and start anew, or discontinue his attempts entirely until he becomes more adept with the technic.

PERIDURAL BLOCK

The peridural space extends from the foramen magnum to the sacrococcygeal junction. The outer wall consists of supportive ligaments of the vertebrae and the periosteum; the inner wall is the dura mater.

Within the space are contained plexuses of venules and arterioles, lymph vessels, and adipose and connective tissues. The presence of a negative pressure within the peridural space is an accepted fact and is used to determine whether or not the point of the needle is in the space. Deposition of anesthetic solutions may be accomplished at the thoracic, lumbar, or caudal areas. This paper is concerned with only the latter two areas.

The patient is positioned, prepared, and draped as for a spinal puncture. I learned to do peridural technic with the patient in the sitting position; hence, for me, this is an easier approach than when the patient is acutely flexed in the lateral recumbent position. Being consistent in repetition of procedures makes the anesthesiologist proficient; therefore, I believe that it is wise to choose a technic and stick with it until it sticks with you.

The materials used in peridural block are identical with those used in placing a catheter within the subarachnoid space except that a short, blunt bevel, 16 gauge Tuohy needle is used so that after the ligamentum flavum is pierced, the flexible meninges are likely to be pushed away without perforation should the needle be advanced too far.

The needle is inserted slowly and gradually in the midline to avoid puncturing of meningeal vessels. Slight resistance will be offered by the skin and interspinous ligament. The needle is advanced to the next point of resistance indicating that the ligamentum flavum has been reached. If bone is encountered at this point, the needle should be withdrawn as in spinal puncture and reinserted at a more suitable angle. The stylet is removed and a few drops of sterile saline solution are placed over the hub of the needle. The needle is further advanced through the ligament until a definite "snap" is felt, indicating perforation of the ligamentum flavum. The saline solution will be aspirated into the needle, demonstrating the negative pressure now existing in the peridural space.

Three other methods are used to provide entrance into the peridural space: (1) the attachment of a 2 or 5 cc. sterile syringe containing sterile normal saline to the hub of the needle when the stylet is removed, (2) the attachment of a 20 cc. syringe containing 5 cc. of air, and (3) the addition of a U-tube manometer. When the space is entered, the manometer shows a negative pressure, or if the saline-filled syringe is used, the saline will be aspirated into the needle. I prefer to attach a partially air-filled 20 cc. syringe, advance the needle with my left hand while exerting pressure on the plunger of the syringe with my right hand. To me this gives the most definite evidence that the peridural space has been entered—the plunger will actually be sucked into the barrel of the syringe. The needle must then be gently aspirated for the presence of spinal fluid or blood.

After it has been determined that the needle is properly placed in the peridural space, a test dose of 5 cc. of the anesthetic solution is injected through the needle and a period of 5 to 10 minutes is allowed to elapse. During this time the catheter is inserted either caudalward or cephalad by directing the Huber point in the desired direction. This waiting period serves as a safety measure to be certain that the catheter has not inadvertently been advanced through the dura where a massive dose of the solution would cause a fatal spinal block. The test dose also helps to make the passage of the catheter more comfortable. The needle is withdrawn and the catheter is secured to the patient in the manner previously described, the distal end being connected to the reservoir of anesthetic solution.

Sympathetic paralysis will be almost as intense with peridural as with intrathecal analgesia; the drop in blood pressure with equivalent heights of analgesia will be about the same as with spinal analgesia, and peridural has the advantages of infrequent sequelae, minimal intercostal paralysis, and lack of complete respiratory paralysis with too high a level of analgesia.

CAUDAL BLOCK

Anatomically the caudal canal is the downward extension of the peridural space. The patient is placed in either the prone or lateral recumbent position, and after proper preparation and anesthetization of the skin, a 16 gauge Tuohy needle with a Huber point or a 15 gauge Love-Barker needle is inserted through the superficial posterior sacrococcygeal ligament into the caudal canal as in the usual caudal technic. Then a catheter is advanced through the needle up into the peridural space to the appropriate level. Progress of the catheter cephalward may be impeded if the catheter twists upon itself or catches on a nerve root or bony irregularity of the sacral canal. Dural puncture is not likely in this region since the dura usually extends no farther caudalward than the second sacral vertebra; however, the usual precautions must be taken to prevent subarachnoid instillation of anesthetic solution in large doses. Should the dura be tapped, it is best to continue the block from this region as a continuous spinal.

The danger of epidural infection with abscess formation is always present since the caudal catheter must be left in place for long periods in a relatively contaminated area. To aid in preventing this complication a generous dab of Sulfonamid creme is placed around the catheter as it emerges from the skin. Sterile sponges again prevent kinking and serve to pad the catheter. Adhesive strips are used to secure the catheter to the patient's back, letting it come to his side at some convenient level. Attachment is made to an anesthetic reservoir or a slow, continuous drip may be used by placing the bottle of solution well above the patient for gravity feed.

DIAGNOSTIC CONDUCTION ANALGESIA

Diagnostic conduction analgesia can extend into any realm the anesthesiologist wishes. Since tapping the dura always subjects the patient to a possible post-dural-puncture cephalalgia, and since most diagnostic procedures of this nature seldom require blocking of motor nerves, it is my opinion that, whenever possible, either the lumbar or sacral peridural routes should be utilized. Because diagnostic procedures entail a relatively long period to be carried out their technics should, if possible, be instituted in the recovery room, anesthesia office, or induction room, or some part of the surgical suite where emergency resuscitative measures are close at hand.

Intractable pain following amputation presents an almost unbearable problem to the patient and a great decision for the surgeon. In order to block vasomotor, visceromotor, and stretch fibers, the continuous spinal method is the one of choice in this instance. Large amounts of 0.2 per cent procaine are used to procure the block. By this method differentiation can be made between pain of local origin and that projected from sensory areas of the cortex. If local pain is obliterated, the patients can be benefited by additional surgery;

other patients cannot. Intractable pelvic pain can also be obliterated; this procedure serves as a quick, temporary means of relief until localized nerve block can be established.

Conduction analgesia can be used in detection of the malingerer. Imagined pain will not be relieved no matter how high an analgesic level is established, but actual pain will be definitely relieved, allowing the patient to assist the clinician in the localization of symptoms.

CONTINUOUS CONDUCTION ANALGESIA IN ECLAMPSIA

Perhaps the most gratifying uses of continuous conduction analgesia, and the one for which I am most frequently called upon, is in the management of eclampsia. Spasm of the terminal arterioles throughout the entire body, especially marked in the kidneys, is believed to be the pathologic process occurring in this condition. Treatment, therefore, must be aimed at the angiospastic mechanisms. It appears rational to utilize conduction analgesia in the management of eclampsia, as in the treatment of ischemia occurring in other pathologic conditions.

Blocking efferent impulses from the sympathetic nerves to the lower extremities results primarily in vasodilatation in that area and increased urinary output because of the relaxing effect upon the smooth muscles of the renal pelvis and ureters, and upon the vascular bed of the kidney itself. The amount of epinephrine excreted by the adrenal glands is decreased. The consequent relief of hypertension eases the load carried by the heart, relieves pulmonary edema, increases cerebral oxygenation, relieves headache and visual disturbances, and decreases peripheral edema.

Blocking the motor nerves to the skeletal muscles decreases restlessness and venous return because of muscular relaxation and peripheral vasodilatation. This, in addition to the relief of pain, increased cerebral oxygenation and decreased blood pressure, lessens the nervous irritability and convulsions which the patient experiences.

Although some authorities[3,4] prefer continuous caudal or lumbar peridural blocks in the management of eclamptic patients, it has been my experience and that of my co-workers in obstetrics that more satisfactory results have been generally obtained when continuous spinal analgesia by the catheter technic was instituted. With the use of the continuous spinal, more lasting and profound reductions in blood pressure and greater increase in urinary output have been obtainable than with either the continuous caudal or lumbar peridural routes. In order to achieve marked relief of eclamptic symptoms it is essential that the level of analgesia be maintained sufficiently high (lower thoracic nerves) to relieve renal ischemia.

Frequent and accurate checking of blood pressure must be carried out in order to prevent a precipitous fall with resultant peripherovascular collapse, shock, and consequent embarrassment of fetal heart tones. Since the decrease in systolic blood pressure will be proportionately greater than the decrease in diastolic blood pressure, care must be taken to maintain a pulse pressure capable of insuring adequate circulation, particularly of the heart muscle.

In order to prevent local infection at the site of emergence of the catheter, as well as to minimize pulmonary complications, massive doses of intravenous penicillin should be given routinely throughout the period of conduction anesthesia.

A relatively nontoxic, dilute agent is preferable in these cases because more essential than motor nerve block is sympathetic and sensory nerve block. However, it is most important that the agent used be familiar to the anesthesiologist, for this will enable him to get more satisfactory results than if he uses one experimentally.

CASE HISTORY

Just 2 days after I had assumed duties at the Houston Osteopathic Hospital I was confronted with a severely toxic 24-year-old primigravida with pitting edema of the entire body, at 4 plus albuminuria, systolic blood pressure of 190, diastolic blood pressure of 120, at term with a fetus in posterior occiput presentation. Since the hospital did not possess continuous spinal equipment, I attempted to do a single dose spinal, but was unable to tap the dura because of the short spinal needle with which I had to work and the obesity of the patient (196 pounds, 5 feet, 3 inches). Much to my amazement I was able to insert a caudal needle. Using a borrowed continuous caudal set and the 1 per cent procaine and 1.5 per cent Monocaine available, I maintained the blood pressure at 150-160/110 by repeated injections of 10-20 cc. of solution every 30-60 minutes. The level of analgesia stayed at approximately 2 fingerbreadths below the umbilicus during this time, and the fetal heart tones remained consistently at 160 per minute.

The next day being Sunday, I created a city-wide disturbance by attempting to secure 1.5 per cent Metycaine. By noon I had three 200 cc. bottles of Metycaine —the entire supply in the city of Houston! Average doses of 15 cc. of Metycaine at approximately hourly intervals maintained the blood pressure as before. During this time an attempt was made to induce labor with 1:2,000 intravenous Pitocin with no results.

By 6:00 p.m. Monday, the third day of conduction analgesia, a surgical supply house had secured a long 16 gauge Tuohy needle and vinylite catheter from out of town, and I was able to institute continuous spinal analgesia at approximately the level of the third and fourth lumbar vertebrae. At this time 4 mg. of subarachnoid hyperbaric Nupercaine caused the blood pressure to drop to 88/70, and the fetal heart tones to become irregular and weak. Intranasal oxygen at 5 liters per minute was started immediately, and 5 mg. of Vasoxyl intravenously restored the blood pressure to 100/70 almost immediately. Thereafter, I administered 1 cc. (15 mg.) of 1.5 per cent Metycaine every hour, and the blood pressure stabilized again at 130-146/100-110, with the level of analgesia at 2 fingerbreadths below the umbilicus.

An attempt to induce labor was again made during the evening; no cervical dilatation and only fair uterine contractions resulted, but irregular fetal heart tones were produced. Upon surgical consultation it was decided to perform a cesarean section at 11:30 p.m.

When the patient was brought to surgery I was unable to determine the level of analgesia because she had become very restless and somewhat irrational. Since the last 15 mg. dose of Metycaine had been given 30 minutes earlier, I arbitrarily introduced 50 mg. of procaine through the catheter, placing the patient in approximately a 10 degrees Trendelenburg position. The blood pressure then dropped to 100/70 with no subjective symptoms. A viable female infant

was delivered at 11:54 p.m. A total of 200 mg. of Pentothal Sodium and minimum cyclopropane-helium-oxygen mixture was given for restlessness until surgery terminated at 12:35 a.m. The final blood pressure recorded in surgery was 140/86. The spinal catheter was left in place, capped and taped to the patient's side where it remained for the next 3 days. During this time her blood pressure remained at 140-160/86-110, and no additional injections were given through the spinal catheter. Urinary output was adequate.

The patient had a very stormy surgical recovery, due in part to a rather marked hemorrhage during surgery. However, she was discharged from the hospital on the seventeenth postoperative day in good condition. Her last recorded blood pressure was 138/80.

Last January at Detroit Osteopathic Hospital a fractional spinal sympathetic block was run for 1 week and 2 hours on an eclamptic patient 8 months pregnant, with convulsions, oliguria, and marked hypertension. A vinylite catheter was inserted at the third and fourth lumbar interspace and directed cephalad. During this time a total of sixty-four injections were made consisting of 80 mg. of hyperbaric Nupercaine, 40 mg. of 1 per cent Pontocaine, and 1,020 mg. (68 cc.) of Metycaine. In this case convulsions were not controlled by Metycaine, but were controlled by hyperbaric Nupercaine.

Cesarean section was withheld to get the patient in optimum condition and also to give the baby a better chance of survival. The catheter was left in place for 2 days after the section at which a living premature female infant was delivered. An attempt to discontinue the sympathetic block was made, but the patient again had convulsions. The interval between injections was gradually lengthened until the patient was symptom-free.

In the above case Veriloid was ineffective in controlling the symptoms. However, a combination of fractional spinal anesthesia and Veriloid effectively controlled hypertension in a 16-year-old postpartum eclamptic patient in whom it was impossible to control the hypertension with any drug via the spinal route.

Stander[5] in his experiments on dogs found an increase in uric acid and blood sugar, inorganic phosphorus, and lactic acid with a concurrent decrease in the carbon-dioxide combining power when a general anesthetic agent such as ether was used. These changes parallel those found in the eclamptic patient. Thus it can be seen how general anesthetic agents, rather than surgery itself, increase the morbidity and mortality of the eclamptic and pre-eclamptic. It can also be seen how imperative it is that some form of conduction anesthesia be used when radical treatment of eclampsia is indicated.

CONDUCTION ANALGESIA IN PERIPHERAL VASCULAR DISEASE

It has been shown that the vasoconstrictor fibers to the blood vessels of the skin of the lower limbs leave the spinal cord by way of the white rami and terminate in the ganglia of the sympathetic trunk. From here postganglionic fibers arise, pass through the gray rami to all the spinal nerves, and are distributed with their cutaneous branches, ultimately leaving these branches to join all the vessels including the capillaries.

Sympathetic nerves to the extremity, then, are vasoconstrictor in type. Normally, systemic temperature is in part regulated by a greater or lesser degree of vasoconstriction of the peripheral vessels—a condition which likewise exists in patients whose circulation is impaired by peripherovascular disease. Interruption of the activity of the sympathetic nerves supplying the extremity will greatly increase the circulation to the part by release of these vasoconstrictor impulses—the principle of modern peripherovascular disease therapy.

Lumbar sympathetic block may be helpful in determining the element of spasm in any ischemic condition of the lower extremity and may be used to give relief from the intermittent claudication resulting from such spasm. For thrombophlebitis of the lower extremity it should be resorted to as soon as the diagnosis has been established. It is also used in an attempt to lower the line of demarcation and avert gangrene following arterial occlusion. It has been used to relieve the pain of frostbite gangrene and has found favor not only in obliterating the pain, but of aiding in the healing of tropical ulcers and the ulcers of peripherovascular gangrene and diabetes. In the latter instance, of course, the initial problem of diabetic management must first be remedied before healing can progress toward permanent result.

Clinical investigation concerned with peripheral circulation depends largely on skin temperature changes that occur when the vasomotor nerves are blocked. These changes can be minutely measured with a McKesson Dermalor or a thermocouple thermometer.

When repeated lumbar sympathetic nerve block is necessary, a simpler and more certain method of its production than that afforded by paravertebral injection of the ganglionic chain or sciatic nerve must be used because some patients will not tolerate a repetition of a procedure which is often painful and time-consuming. Anyone who is experienced in placing a catheter in the epidural space through the sacral hiatus and has adequate personnel to carry out a continuous block in this manner has, with few contraindications, a more certain and more pleasant means of giving relief to the victim of peripherovascular disease. Obviously this method is contraindicated, as is any caudal analgesia, in the presence of marked obesity, skin infections about the area of catheter insertion, deformities of the sacrum, low-lying dura, disorientation of the patient, or where urinary or fecal incontinence adds to the already existing danger of infection.

While my experience in the treatment of peripherovascular diseases by the continuous caudal route has been to use 1.5 per cent Metycaine or 0.15 per cent Pontocaine in repeated doses of 5-10 cc. once a satisfactory block has been established, many writers[6,7] advocate the use of very dilute solutions (0.02-0.05 per cent) of Pontocaine or procaine. With these concentrations relief of pain has been obtained without disturbing bowel and bladder function.

OTHER DIAGNOSTIC AND THERAPEUTIC APPLICATIONS OF CONDUCTION ANALGESIA

Various tests have been used to predict which hypertensive patients will benefit from sympathectomy. Intermittent intrathecal injections of procaine (0.2-0.4 per cent) or 1.5 per cent Metycaine by the sacral peridural route should be used in sufficient quantity to obtain a sensory level to the seventh dorsal vertebra or the xiphoid. The extent of sympathetic chain depression can be determined by measurement of electric

skin resistance, or more simply by frequent blood pressure readings over a period of an hour or so following the establishment of the aforementioned skin level. An appreciable fall in blood pressure during this time indicates a favorable prognosis for a sympathectomy. In order to obtain the most exact information from this test, it is wise to inject a large dose (40-50 cc.) of 1.5 per cent Metycaine through the caudal catheter rather rapidly and then maintain the skin sensory level at the seventh dorsal for about an hour with smaller doses (5-10 cc.) of the agent. It must be emphasized that measures to combat profound hypotension and possible reactions to the anesthetic agent should be immediately obtainable.

The underlying factor producing the anuria in lower nephron nephrosis is believed to be a renal cortical ischemia which results from stimulation of the renal vasomotor nerves and diversion of the renal blood flow from cortex to medulla. This mechanism may be prevented or the resulting anuria treated by blocking the renal vasomotor pathways by continuous caudal analgesia.

Spinal analgesia by the continuous route has proved efficacious in treating pulmonary edema resulting from arteriosclerotic and hypertensive heart disease. In these cases pulmonary edema develops because of left ventricular failure with consequent increase of pressure in the pulmonary circuit and extravasation of fluid. Decreased venous return to the heart is accomplished by the ability of spinal analgesia to pool blood in the lower extremities. Since these patients are usually in the middle or old age groups, care must be taken not to disturb their already diseased cardiovascular system by sudden marked periods of hypotension. Small and frequently repeated doses of any spinal anesthetic drug should be given to produce a block involving only the lower lumbar and sacral nerves.

HEADACHE AS A COMPLICATION

Postspinal headaches constitute the most often observed and most vigorously discussed complication of spinal analgesia. Although relatively benign in its effect on the patient's general condition, this complication may be distressing enough to keep an otherwise active patient confined to bed for several days, making impossible any efforts on the part of the surgeon or obstetrician for early ambulation of his patient.

Lumbar puncture headaches have been recognized for many years, and there have been many conflicting theories about their cause and just as many reports and theories about their treatment. While the theory that they are caused by postpuncture leakage of cerebrospinal fluid through the dural rent, with subsequent changes in cerebrospinal fluid dynamics, loss of cushioning effect of the brain, and pain due to pressure or traction on sensitive brain structures and large vessels, is still not universally accepted, it is the most logical and encompassing theory advanced today.

Because of the likelihood of headache reason it is most important that spinal taps for any reason be done by a very systematic routine, and that whenever possible the smallest lumbar puncture needle available be used. An amazing reduction in the incidence of headache can be established by using 24 and 26 gauge needles. Just recently Cappe and Deutsch[8] reported on the lessened incidence of headache which they experienced upon using a cone tipped needle having no cutting edge. Since the dural fibers were split rather than cut, the opening readily sealed itself with little, if any, loss of cerebrospinal fluid. At best, however, luck —if we can call it that—runs out, and we are confronted with a severe persistent headache failing to respond to any form of drug therapy advocated for its relief.

At the Massachusetts Memorial Hospital in Boston[9] a series of cases was run in which the anesthetic solution was introduced into the subarachnoid space, the needle withdrawn into the epidural space, and 10 to 20 cc. of normal saline deposited therein before the needle was completely removed. The efficacy of this method appears to depend on the building up of a head of pressure near the dural rent and preventing leakage of spinal fluid into the epidural space. This prevents a concomitant decrease in cerebrospinal fluid pressure. Although it is not clear whether this saline solution remains in the region long enough to accomplish its mission, it is conceivable that it prevents leakage long enough for a fibrin clot to form or for the dura to seal itself.

This procedure tends to lengthen the period which the busy anesthesiologist takes to prepare his patient for the surgical or obstetrical "prep," and may, theoretically, tend to produce a one-sided block; however, it undoubtedly is very sound physiology and adheres to the old adage, "An ounce of prevention is worth a pound of cure." At the Detroit Osteopathic Hospital we did not follow this routine, but many of us in the Department of Anesthesiology were so encouraged by the apparent decrease in the incidence of cephalalgias following the use of fractional spinal methods that we routinely injected 20 to 30 cc. of normal saline through the spinal catheter before withdrawing it at the termination of surgery.

While a "one-shot" caudal of 40 to 80 cc. of normal saline is often adequate to give dramatic and lasting relief to the sufferer of postlumbar puncture cephalalgia, its greatest disadvantage is the need for repetition on successive days should the headache recur with its original severity. To obviate this somewhat uncomfortable repetition for the patient, an indwelling catheter is placed in the caudal canal. An initial dose of 40 to 50 cc. of normal saline is administered via the catheter, the catheter closed off, and the patient instructed to sit up and move about at will. As a rule the headache is completely gone or at least markedly relieved. The patient is rechecked in an hour or two; if discomfort has returned an additional 30 cc. of saline is injected. On the following day the patient is again seen and questioned regarding any return of his headache. Further saline injections are given if needed. In only the most stubborn cases is it necessary to continue this therapy over a period of several days.

For the cephalalgic patient who is not a caudal candidate, a normal saline lumbar epidural by either single injection or indwelling catheter can be substituted for the caudal route of therapy. This route should be used only as a last resort because of the added risk of inadvertently tapping the dura in this area and thus exaggerating the existing cause of the complaint.

CONTINUOUS INTRASPINAL CATHETER TECHNIC

Repeating the same nerve block numerous times on the same patient has many undesirable features, and has led to the adoption of a modification of the continuous intraspinal catheter technic.

The method used by Sarnoff and Sarnoff[10] is to make a skin wheal, insert a thin-walled 18 gauge needle to the desired point as determined by fluoroscopy or by applying an electrical potential to the insulated nerve block needle when it is believed to be in the proper position, and then to pass through this needle a polyethylene catheter and its stylet to the needle tip. The needle is then gently withdrawn over the tubing, the stylet is removed from the catheter, a blunt 23 gauge needle is inserted into the open end of the tubing, and the anesthetic agent is injected. A needle plug is placed in the needle when the desired block has occurred.

The most difficult step in this procedure is to secure the catheter to the skin. This is accomplished by making a heavy circular coating of collodion on the skin at the point of emergence of the tubing. Narrow strips of adhesive tape wound around the tubing are then fixed to the skin at the edge of the collodion. The remainder of the tubing, the needle, and its stopper are then placed in sterile gauze and taped to the patient's skin.

This has proved a successful method of treating intractable hiccups by continuous phrenic nerve block when conventional therapeutic measures have failed and surgical intervention is deemed inadvisable. Similarly, brachial plexus and stellate ganglion blocks can be made continuous technics: the brachial plexus block being utilized in sympathetic block of the arm and forearm, for the differential localization of pain, and for pain relief in upper extremity bursitis and herpes zoster; the stellate ganglion block for therapeutic relief of the vasospasm of the upper arm, brain, face, and lungs resulting from cerebral accidents and characterized by hemiplegia. It is an aid in arterial dysfunctions such as Raynaud's disease and the arterial spasm followed by gangrene of an extremity if arterial injection of Pentothal Sodium is accidentally made; venous dysfunctions such as thrombophlebitis; arteriovenous dysfunctions such as postmastectomy edema; and post-traumatic dystrophies associated with causalgia, edema, and osteoporosis. The secret of the successful stellate ganglion block in relieving hemiplegia is for the anesthesiologist to be given the opportunity of performing the block within the first 8 hours after the accident has happened. Blocking of the stellate ganglion will also give relief during status asthmaticus and angina and other forms of cardiac pain, and it will cause hyperhidrosis to cease in the upper extremity. It can also be used in foretelling the advisability of stellate ganglionectomy and dorsal sympathectomy.

Since the chemicophysiology of the extensively burned patient is so greatly altered as to make elimination of general anesthetic agents a real problem, it has been the policy of anesthesiologists in the past to use nitrous oxide-oxygen for analgesia and basal narcosis when caring for these patients. Because such frequent anesthetics are demanded for change of dressings and skin-grafting, an added load is placed on the already overworked kidneys and liver when general anesthetic agents are used. Continuous methods can be utilized not only to give surgical anesthesia, but to supply comforting analgesia following surgery without large doses of narcotics. Only very dilute anesthetic solutions are needed to produce sensory block therapy, yet the resulting sympathetic block provides a gentle oozing of fresh blood to the skin-grafted areas to initiate quicker healing.

SPLANCHNIC BLOCK IN PANCREATITIS

Patients having pancreatitis are usually found to be extremely ill, often in such severe pain that even large doses of morphine give them no relief, and, theoretically, morphine may aggravate the condition by increasing the spasm of the sphincter of Oddi. As early as 1945 splanchnic anesthesia was reported to give complete relief of symptoms and, in addition, favorably affect the course of the disease.[11] The theory is that drainage of the pancreatic enzymes into the duodenum is brought about by relaxation of the sphincter of Oddi and that spastic blood vessels become dilated.

In order to obtain a block of the splanchnic nerves, it is necessary to reach the sixth, seventh, eighth, and ninth thoracic segments of the cord. To accomplish this the epidural catheter must be threaded well cephalad; if this is impossible, lowering the patient's head or injecting additional anesthetic solution will raise the level of the block to the necessary segments. Injections are repeated at intervals determined by the requirements of the individual patient.

CONDUCTION ANESTHESIA IN CONTROL OF BLEEDING

In reporting the following I feel that I may be stretching the term "treatment" in regard to continuous conduction analgesia; however, at the Detroit Osteopathic Hospital a particular use of continuous lumbar peridural block was found to be so advantageous that I would like to consider it briefly. It has to do with control of bleeding rather than anesthesia per se.

An absolutely quiet patient and a bloodless field are the prime requisites for the surgeon doing a fenestration operation for otosclerosis. Dr. Lloyd Seyfried felt that he was encountering needless and troublesome bleeding at the operative site. Dr. Maurice Howlett produced a satisfactory solution to this problem by instituting continuous lumbar peridural analgesia with its concomitant lower extremity vasodilatation to cause compensatory vasoconstriction of the operative field. We used 1.5 per cent Metycaine in doses just sufficient to maintain the systolic blood pressure of the average patient at approximately 100 mm. of mercury. Reinforcement of the block every 45 minutes to 1 hour was necessary. Vasopressors, although seldom needed in this instance, should be at hand to combat any profound hypotension.

By the same token, continuous lumbar peridural analgesia can be used to cause compensatory vasoconstriction of the operative field during thyroid surgery. Sympathetic overactivity causing increased adrenal gland secretion is a conspicuous feature of the thyrotoxic patient who cannot be improved to any greater extent by medical means preoperatively. Extremely high pulse and blood pressure levels can be averted, or at least controlled, in patients in thyroid crisis by spinal analgesia to the level of the fourth and fifth thoracic segments. When used as part of the anesthetic procedure (whether it is local or general), beneficial effects are seen in the thyrotoxic patient undergoing surgery, and during the occasional postoperative storm which may ensue.

CONCLUSION

In closing, let us remember that the drugs and technics which the anesthesiologist uses for the above-

mentioned therapeutic measures far outweigh those found in the internist's armamentarium, and their use, like the concepts of osteopathy, is based on scientifically seeking and removing the underlying cause of disorders. Remember, too, never to hesitate to suggest the use of methods of anesthesiology in managing the "problem case." While at first they may be received with skepticism, their results will vindicate their use.

2122 Cheshire Lane

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BOOK NOTICES

(Continued from page 644)

tion reborn in the successive editions, from Macmichael's to those of a new day; a never-failing idealism which has always characterized medicine at its best; and, lastly, a beautiful book.

And for the making and publishing of this example of a modern book at its best, the publisher should have his reward—a goodly sale among all physicians who are appreciative of the many bypaths which characterize their calling.

PLANNING GUIDE FOR RADIOLOGIC INSTALLATIONS. By the Committee on Planning of Radiologic Installations of the Commission on Public Relations of the American College of Radiology. Wendell G. Scott, M.D., Chairman. Cloth. Pp. 336, with illustrations. Price $8.00. The Year Book Publishers, Inc., 200 East Illinois St., Chicago 11, 1953.

This reference manual fills a longfelt need. Granted that an individual knows something of the problems of radiologic installations, he will find them all discussed here, intelligently and practically. The book covers all the problems that will come up eventually in any case of building a radiology department and some of which will be forgotten in the midst of an actual project. One of the book's strongest points is that it is not the ideas of one man, no matter how well informed. It is a joint endeavor, as is pointed out in the preface and confirmed by an examination of the text, of radiologists and of representatives of companies manufacturing x-ray equipment and films, of federal health agencies, of the American Hospital Association, and the American Institute of Architects.

The American College of Radiology should be warmly commended for sponsoring the book and the publishers for making it possible to place the text in the hands of the many who need such a manual and who hitherto have had to consult many sources or have been forced to limit their information to one or two advisors. Chapter 6, devoted to "Pertinent 'Don't's' in Construction or Remodeling of Radiologic Installations," is a new set of commandments—thirty-four of them—not to be violated with impunity. The flexibility of treatment of the subject is shown by the text's apparent usefulness to hospitals of all sizes—radiologic services are now available in almost every hospital of fifty-bed capacity or more in the United States, and many smaller institutions have a department manned by a radiologist on a part-time basis. This is a book with which radiologists, hospital superintendents, and all persons having any responsibility for an indispensable service of medicine should be acquainted.

PRIVATE WORLD OF PAIN. By Grace Stuart, with an appendix by John Malins, M.B., M.R.C.P., Assistant Physician to the Birmingham United Hospitals. Cloth. Pp. 191. Price $2.50. Distributed by The Macmillan Company, 60 Fifth Ave., New York, 1953.

Of the making of "uplift" books there is no end; rarely are they other than dull and somewhat presumptuous, and less often does it seem that they could be effective in helping people who sorely need to find—as it is termed by this author—an "alternative world." But this book is different, wisely and wonderfully. Grace Stuart was stricken with rheumatoid arthritis more than 30 years ago—midway of a brilliant university career. She now belongs to that group of patients for whom cortisone was a tremendous aid, and about that her physician has written a useful appendix. But she never would have made the response to the drug that she did, had she not kept herself living through the years to a fulness seldom reached by individuals who have no disability. How she did it, in spite of continuous pain and inescapable limitation, and with an entire realism, is left for the reader to experience. It remains as a study of the wonder of human spirit when its full capacity is utilized.

This is a book for all doctors to read well, the better to appreciate the quality of fabric with which they deal, and then to prescribe it to patients living within a shadow to read slowly and carefully and very thoughtfully—and then to think over. One wonders if any person of average literacy might not gain new reasons for living from reading the book's less than 200 pages, simply and beautifully written. In any event this little volume, published in England in 1953, should be on the prescription list of every doctor, for books can be healing agents, and often of avail when drugs are not.

A FORMULARY FOR EXTERNAL THERAPY OF THE SKIN. By Chester N. Frazier, M.D., Dr. P.H., Edward Wigglesworth Professor of Dermatology, Harvard Medical School; Chief of the Dermatological Service, Massachusetts General Hospital, Boston, Massachusetts, and Irvin H. Blank, Ph.D., Research Associate in Dermatology, Harvard Medical School, Massachusetts General Hospital, Boston, Massachusetts Cloth. Pp. 118, with illustrations. Price $3.25. Charles C Thomas, Publisher, 301-327 East Lawrence Ave., Springfield, Ill., 1954.

This formulary is a series of lectures that explain the reasoning that should be followed in dermatologic prescription writing. Great stress is placed on a sound and logical understanding of what one is trying to accomplish. Brief historical background is given, and care of skin is rationally discussed.

Physiology of skin and the basic principles of the effect of lotions, ointments, and pastes, along with the chemical and physical characteristics of each, are well covered. Emollients and protectives and their chemistry and uses are the subject of a chapter.

In the preparation of formulas the manual falls short. Except in large institutions, with special pharmacy available, the prescriptions suggested here would often be difficult to have compounded. Hexachlorophene is mentioned in a number of connections. The authors recommend its incorporation in antipruritic lotions and ointments. It is used in their antiinfectious lotions and ointments, and it is suggested as a desirable chemical in some soaps. Antibiotics are minimized since the formulary is devoted to external therapy.

The manual is excellent background reading, and is recommended for its basic principles and their application to dermatologic therapy.

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Conventions and Meetings

Announcements

American Osteopathic Association, Fifty-Eighth Annual Convention, Royal York and King Edward Hotels, Toronto, Canada, July 12-16, inclusive. Program Chairman, Campbell A. Ward, 51 Cass Ave., Mt. Clemens, Mich.

American College of Osteopathic Internists, annual meeting, Philadelphia, October 7-10. Secretary, Glennard E. Lahrson, 460 Staten Ave., Oakland 10, Calif.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Jacksonville, Fla., February 7-10. Secretary, Harold K. Morgan, 3268 W. 32nd Ave., Denver 11.

American College of Osteopathic Surgeons, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Administrative Secretary, Mrs. O. F. Martin, Box 474, Coral Gables 34, Fla. Program Chairman, J. Donald Sheets, 12561 Third Ave., Highland Park 3, Mich.

American Osteopathic Academy of Orthopedics, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Secretary, J. Paul Leonard, 2673 W. Grand Blvd., Detroit 8. Program Chairman, Walter R. Garard, 809 S. Hobart Blvd., Los Angeles 5.

American Osteopathic College of Anesthesiologists, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Secretary, Crawford M. Esterline, Box 155, Kirksville, Mo. Program Chairman, Armin L. Karbach, 316 N. Center, Arlington, Tex.

American Osteopathic College of Radiology, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Secretary, H. Miles Snyder, Art Centre Hospital, 5435 Woodward Ave., Detroit 2. Program Chairman, John H. Pulker, Flint Osteopathic Hospital, 416 W. Fourth Ave., Flint 4, Mich.

American Osteopathic Hospital Association, annual meeting, Hotel Baker, Dallas, Tex., October 31-November 4. Executive Secretary, Mr. Robert P. Chapman, 1011 Kahl Bldg., 326 Third St., Davenport, Iowa.

Canada, annual meeting, King Edward Hotel, Toronto, September 16-18. Secretary, Miss Joyce S. Currie, 609 Medical Arts Bldg., Montreal 25. Program Chairman, A. E. Wilkinson, 616 Medical Arts Bldg., Montreal 25.

Central States Society of Proctology, Van Orman Hotel, Fort Wayne, Ind., October 17-19. Secretary, Ralph Deger, 1121 Xenia Ave., Dayton, Ohio.

Colorado: See Rocky Mountain Osteopathic Conference.

Hawaii, annual meeting, Honolulu, October 25-27. Secretary, Joseph W. Stella, 38 Young Hotel Bldg., Honolulu 9. Program Chairman, C. W. Wyman, 417 National Bldg., Honolulu 13.

Indiana, annual meeting, French Lick Springs Hotel, French Lick Springs, May 14-17. Secretary, Arabelle B. Wolf, 809-13 Odd Fellows Bldg., Indianapolis 4.

Kentucky, annual meeting, Brown Hotel, Louisville, September 29-30. Secretary, Harold D. Benteen, 2048 Winchester Ave., Ashland. Program Chairman, Martha Garnett, 417 Fincastle Bldg., Louisville 2.

Michigan, annual meeting, Civic Auditorium, Grand Rapids, October 4-6. Secretary, P. Ralph Morehouse, 214 S. Superior St., Albion. Program Chairman, B. F. Dickinson, 222 E. Sixth St., Royal Oak.

Missouri, annual meeting, Hotel Colonial and Kentwood Arms Hotel, Springfield, October 13-15. Executive Secretary, Mr. Paul D. Adams, 325 E. McCarty St., Jefferson City. Program Chairman, L. R. Morgan, 3014 S. Main St., Joplin.

New Mexico, annual meeting, Albuquerque, April 28-30. Secretary, Robert E. Smith, 205 N. First St., Lovington.

New York, annual meeting, Hotel Syracuse, Syracuse, October 15-16. Secretary, Robert E. Cole, 417 S. Main St.,

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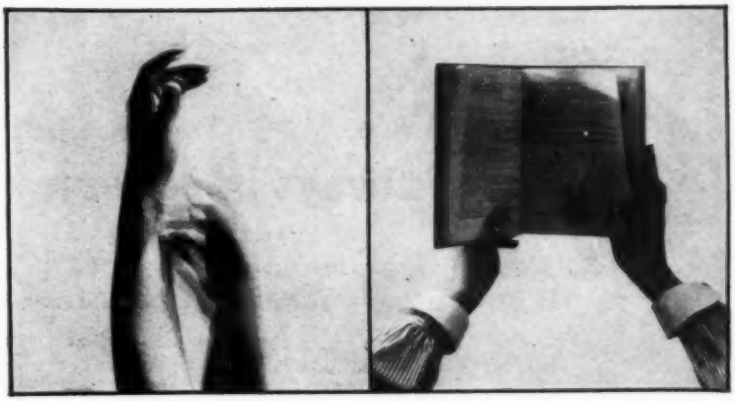

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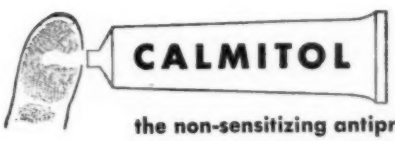

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North Carolina, annual meeting, Battery Park Hotel, Asheville, October 21-23. Secretary, S. Dales Foster, 710 Public Service Bldg., Asheville. Program Chairman, Elizabeth E. Smith, 18 Wall St., Asheville.

Oklahoma, annual meeting, Biltmore Hotel, Oklahoma City, November 8-10. Executive Secretary, Mr. Walter L. Gray, 210-212 Braniff Bldg., Oklahoma City. Program Chairman, Fred Green, Alva Osteopathic Hospital, 619 Center St., Alva.

Pennsylvania, annual meeting, Penn Harris Hotel, Harrisburg, September 24-26. Executive Secretary, Mr. George W. Thomas, 510 N. Third St., Harrisburg. Program Chairman, Harry E. Binder, 7247 Charles St., Mayfair, Philadelphia 35.

Rocky Mountain Osteopathic Conference, midyear meeting, Broadmoor Hotel, Colorado Springs, Colo., November 12-14. Secretary, C. Robert Starks, 1459 Ogden St., Denver 18.

Western States Osteopathic Society of Proctology, annual meeting, Shamrock Hotel, Houston, Tex., October 4-6. Secretary, E. F. Walters, 925 E. South Temple St., Salt Lake City 2.

State and National Boards

ALBERTA

Examinations in September at Edmonton. Address Mr. G. B. Taylor, secretary, Medical Board, Office of the Registrar, University of Alberta, Edmonton.

ARIZONA

Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

Basic science examinations September 21 at the University of Arizona, Tucson. Address Herbert D. Rhodes, Ph.D., secretary, Basic Science Board, University of Arizona, Tucson.

COLORADO

Basic science examinations in September. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver 18.

CONNECTICUT

Basic science examinations October 9 at New Haven. Applications must be filed 2 weeks prior to examination. Address Mr. Herbert S. Harned, secretary, Board of Healing Arts, 52 Whitney Ave., New Haven 10.

DISTRICT OF COLUMBIA

Basic science examinations in October. Address Mr. Paul Foley, Deputy Director, Department of Occupations and Professions, 1740 Massachusetts Ave., N.W., Washington 6.

HAWAII

Examination dates by Territorial Law are usually the first Wednesday, Thursday, and Friday of January, April, July, and October. One of these dates is set for examination after approval of candidate's application by the Board. Address Frank O. Gladding, D.O., secretary, Board of Osteopathic Examiners, 504 Kauikeolani Bldg., Honolulu 13.

ILLINOIS

Examinations October 5-7, 160 N. La Salle St., Chicago. Address Mr. Frederic B. Selcke, Superintendent of Registration, Department of Registration and Education, State House, Springfield.

IOWA

Basic science examinations October 12 at Des Moines. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

MARYLAND

Examinations in October. Address Christopher L. Ginn, D.O., secretary, Board of Osteopathic Examiners, 419 N. Charles St., Baltimore 1.

MICHIGAN

Basic science examinations in October. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, 410 W. Allegan, Lansing.

MINNESOTA

Basic science examinations October 5-6, 101 Wesbrook Hall, University Campus, Minneapolis. Applications must be filed by September 10. Address Raymond N. Bieter, M.D., secretary, Board of Examiners in the Basic Sciences, 105 Millard Hall, University of Minnesota, Minneapolis 14.

MONTANA

Examinations in September. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

NEBRASKA

Basic science examinations October 5-6. Address Mr. Husted K. Watson, Director, Bureau of Examining Boards, Department of Health, State Capitol Bldg., Lincoln 9.

NEVADA

Basic science examinations October 5. Address Donald G. Cooney, Ph.D., secretary, Board of Examiners in the Basic Sciences, Box 9005, University Station, Reno.

NEW HAMPSHIRE

Examinations September 8-9 at Concord. Address John S. Wheeler, M.D., secretary, Board of Registration in Medicine, State House, Concord.

NEW MEXICO

Basic science examinations July 18. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P.O. Box 1522, Santa Fe.

OREGON

Basic science examinations September 11 at Portland. Address Mr. John R. Richards, secretary, State Board of Higher Education, Eugene.

RHODE ISLAND

Professional examinations October 7-8. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

Basic science examinations August 31. Address Mr. Casey.

SOUTH DAKOTA

Examinations July 20-21 at Sioux Falls. Address Mr. John C. Foster, executive secretary, Board of Medical and Osteopathic Examiners, Room 300, First National Bank Bldg., Sioux Falls.

TENNESSEE

Examinations in February and July at Nashville. Applications must be filed 15 days prior to examination. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

WISCONSIN

Basic science examinations September 24 at the Assembly Chamber, State Capitol, Madison. Applications must be filed by September 16. Address Mr. William H. Barber, secretary, Board of Examiners in the Basic Sciences, 621 Ransom St., Ripon.

WYOMING

Examinations October 4. Address Franklin D. Yoder, M.D., secretary, Board of Medical Examiners, New State Office Bldg., Cheyenne.

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6. Brewd, Victor L.: Rehabilitation of Hearing, 1950.
7. Kugelmass, I. Newton: Handbook of the Common Acute Infectious Diseases, 1949.

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September 1—Ohio, $2.00. Address H. M. Platter, M.D., secretary, State Medical Board, Wyandotte Bldg., Columbus 15.

September—Nebraska, $2.00. Address Mr. Husted K. Watson, director, Bureau of Examining Boards, State Department of Health, Lincoln 9.

EXAMINATION BY NATIONAL BOARD

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

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1. Reich, C., and Mulinos, M. G., Treatment of Refractory Nutritional Anemia with Gelatine. Bull. N. Y. Med. Coll. March 1953.

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Part III is an oral and practical examination given in Philadelphia, Chicago, Kirksville, and Los Angeles under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles, therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

These are oral examinations which the candidate may take after having satisfactorily completed the first 6 months of a 1-year internship in a hospital approved by the American Osteopathic Association for intern training. Part III is given annually at the above-named colleges.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1-year internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.

THE GOLD-HEADED CANE IN THE LABORATORY*

By Rene J. Dubos, M.D., Sc.D., Ph.D.†

Although most of medical science is of very recent acquisition, the healing art has been practiced effectively for thousands of years without the benefit of laboratory knowledge. The skill of ancient physicians was largely empirical, but it permitted them, nevertheless, to formulate prognoses, relieve symptoms, and not uncommonly effect cures. It was an empiricism based on wisdom, experience, and knowledge of human nature.

The fashionable English physician of the late 17th century went on his sick calls dressed in a silk coat, breeches and stockings, with buckled shoes, lace ruffles, full-bottomed wig, and carrying a gold-headed cane. Typical of this class was John Radcliffe (1650-1714), physician to William and Mary and to Queen Anne.

EMPIRICISM VS. EXPERIMENTAL SCIENCE

For 150 years, Radcliffe's gold-headed cane was passed along in succession to some of the most successful practitioners of England. In Matthew Baillie (1761-1823), however, it found a master of a different mentality. With a few of his contemporaries, Baillie considered that science was rendering obsolete the assumed gravity and pompous airs of fashionable physicians. Indeed, it was obvious that the clinical art had then come to represent a formal tradition rather than an attitude based on living experience with disease. No longer regarding the gold-headed cane as a necessary appendage of the medical profession, Baillie discontinued carrying it on his sick calls. After his death, his wife found the cane in a corner of the consulting room and presented it in 1825

*Reprinted from *Public Health Reports*, April, 1954.

†Dr. Dubos, member of the Rockefeller Institute for Medical Research, presented the third R. E. Dyer lecture October 22, 1953. The major portion of the lecture is printed here. The full text will be published by the National Institutes of Health. Dr. Dubos enjoys wide recognition not only for fundamental work in bacteriology but also as an author. He pioneered in the study of antibacterial enzymes and antibiotics and isolated tyrothricin and gramicidin, the first crystalline antibacterial substance, from spore-forming bacteria. Another contribution was the development of new media for the cultivation of the tubercle bacillus.

The R. E. Dyer lecture, honoring Dr. Rolla E. Dyer, was established in 1950 and is administered by the National Institutes of Health, Public Health Service.

to the museum of the Royal Academy of Physicians. There it has remained, among other relics of that learned body, a symbol of a phase of medicine without relevance to medical practice or science in the modern era. As the cane went to its final sanctuary, persons who were to change the course of medicine were beginning their lives — Florence Nightingale in 1820, Rudolph Virchow in 1821, Pasteur in 1822, Lister in 1827.

Many of the most brilliant practitioners of clinical medicine of the 19th century, however, continued to hold that their art had nothing of importance to learn from biological sciences. Some of them in particular achieved a place in history by the passion with which they opposed the germ theory of disease. The French clinician Pidoux, a representative of traditional medicine—always impeccably clad in a gold-buttoned coat —was one of those who took up the cudgels against Pasteur at the Paris Academy of Medicine. Where Pasteur saw disease as caused by specific kinds of microbes, Pidoux invoked the concept of diathesis, emphasizing that disease was determined by the particular constitution of the patient. He held the view that any disease could be caused by a multiplicity of external and internal causes and could not be regarded as due to one single specific agent. Necrotic processes could take place "along a number of roads each of which the physician should endeavor to close."

Pidoux and his like spoke in broad clinical terms, basing their doctrines on the phenomena of disease as observed in man. But their vague arguments were no match for the precise experimentation by which Pasteur, Koch, and their followers defended the doctrine of specific causation of infectious disease. Experimental science triumphed over clinical experience, and within a decade the theory of specific etiology of disease was all but universally accepted, soon becoming the dominant force in medicine. Eventually, the doctrine of specificity was generalized to encompass the concept of specific "biochemical lesion" which is proving today as fruitful in the fields of metabolic, degenerative, and neoplastic diseases as the concept of specific microbial etiology has proved in dealing with the problem of infectious diseases. There is no doubt that the doctrine of specific etiology has constituted an instrument of unmatched power for the experimental study of pathological processes and has been responsible for most of the great advances, theoretical and practical, realized in medicine during the past century.

It is now apparent, however, that the concept of single etiology often fails to provide a complete explanation for the pathogenesis of diseases under natural conditions. In "complex infections," two or more microbial agents are required to reproduce the pathological picture—the participation of influenza virus and *Hemophilus influenza suis* in swine influenza being the classical example. In practically all infectious and metabolic disorders, physiological and environmental factors can readily be shown to be important determinants of the disease process.

TUBERCULOSIS INFECTION VS. THE DISEASE

In general, the accounts of the controversies which heralded the germ theory of disease dismiss the views of those who objected to some of its extreme tenets as the voice of obscurantism and reaction in medicine. Yet there were shrewd thinkers among the physicians who were not convinced that micro-organisms alone could account for the causation of disease. They did not deny that micro-organisms were present in diseased tissue. They emphasized rather that micro-organisms could invade and cause disease only after the tissues had been weakened by some form of physiological misery.

Rudolf Virchow was one of those who believed that micro-organisms are secondary invaders and that physiological disturbances are always the primary cause of disease. In 1847, while still a young man of 26, he had served as a member of a medical commission sent by the Prussian Government to investigate the widespread occurrence of epidemics in the industrial sections of upper Silesia. There he noticed that fevers were particularly prevalent among those living under miserable circumstances, and he became convinced that poverty was the real breeder of disease. He pointed out, furthermore, that Negroes

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*Sherber, D. A.: The Control of Bleeding, Am. J. Surg. *86*:331 (Sept.) 1953.

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living in Europe were much more likely to acquire tuberculosis than those who had stayed in their native lands or than white persons of the same age working at the same trades and exposed to the same risks of infection. Did not this incriminate the physiological disturbance brought about by the change from the tropical to the northern climes as primary cause of the disease?

Although Virchow recognized the importance of Koch's discovery of the tubercle bacillus, he had good epidemiological reasons to remain unimpressed while presiding at the now famous meeting of the Berlin Physiological Society in 1882 where Koch first presented his report. Indeed, Virchow would have been even less impressed had he known, as we know today, that almost every adult individual in Europe at that time was infected with virulent tubercle bacilli and that Koch himself was certainly infected, as indicated by the violence of his reaction when he injected tuberculin into his own arm in 1891. Virchow could have argued that since the tubercle bacillus was an ubiquitous component of the environment, the factors which converted mere infection into overt symptoms and destructive pathological changes were the real determinative causes of tuberculosis.

The knowledge accumulated during the past 75 years has left unsolved many of the problems of the pathogenesis of tuberculosis. Tuberculin tests reveal that even in our communities a very large percentage of the adult population has been at some time infected with tubercle bacilli. Yet, the morbidity and mortality of tuberculosis have decreased by ten- to twenty-fold during the past century. It is obvious, therefore, that while the tubercle bacillus is the specific etiological agent of infection, there are other factors which are responsible for converting infection into tuberculous disease. In other words, the etiology of disease cannot be explained entirely in terms of the etiology of infection.

MULTIPLE ETIOLOGY IN HUMAN DISEASE

Another example illustrates the contrast between infection and disease. In man, the herpes simplex virus is usually acquired during early years. Throughout most of the life of the infected individual, the virus lies latent in the body, without causing any symptom or obvious pathology until "provoked" into activity by some physiological disturbance. As is well known, fever (herpetic) blisters can be elicited by a variety of nonspecific unrelated stimuli, as different one from the other as menstruation, colds and fevers of various origin, ultraviolet radiation, or eating cheese. Herpetic blisters thus provide a striking example of an infectious disease of man in which, contrary to the original tenets of the germ theory, the living agent of the disease (the germ) may be present all the time in the host—be intrinsic so to speak—whereas the determinant of the pathological process is some physiological disturbance or some other extrinsic factor of the physicochemical environment.

There are many examples of infections caused by either protozoa, fungi, bacteria, rickettsia, or viruses which are ubiquitous in their distribution among human, animal, or plant communities but which remain in a latent, essentially inactive state under ordinary "normal" circumstances. These latent infections express themselves in the form of disease only after some physiological or environmental factor has caused a primary disturbance which allows the microbial agent to manifest its potential pathogenicity. In fact, the nature of the pathological processes may to a large extent be independent of the specific microbial agent associated with it. Thus, it has been frequently observed during recent years that the intensive use of chemotherapy in the treatment of subacute endocarditis caused by green streptococci, while successful in eliminating these organisms from the lesions and blood stream, often results in their substitution by other microbial species. As a consequence, forms of endocarditis associated with staphylococci or even gram negative bacilli are not uncommon in patients treated with antistreptococcal drugs. Seventy-five years ago, this would have appeared to the opponents of the germ theory as just one more example of a situation in which the fundamental disease is the organic lesion (on the heart valves) and the microbial agents

merely opportunistic secondary invaders.

It is known that most, if not all human beings can acquire bacillary dysentery when first exposed to the disease under the proper field conditions, as shown by the fact that new bodies of troops transported to areas where dysentery is endemic rapidly fall prey to the disease. Yet, despite all the available knowledge concerning dysentery bacilli and despite the overwhelming evidence of the communicability of the disease, it has proved extremely difficult to establish experimentally in man or in animals an infection presenting the typical symptomatic and pathological characteristics of bacillary dysentery.

A few years ago, human volunteers ingested dysentery bacilli of the Flexner group recently isolated from patients and known to be virulent for mice. All precautions were taken to favor the establishment of the infection. It was first determined that the volunteers were not carriers of *Shigella* and had negative histories of diarrheal disease. They were given paregoric and sodium bicarbonate before and after receiving the infective dose, and water in order to dilute the gastric secretions and wash the material through the stomach quickly. Yet, administration of up to 95 billion bacilli failed to produce significant symptoms of dysentery in most volunteers, even though the bacilli could be recovered from their stools on several successive days. No greater success was achieved by introducing along with the bacilli a gelatin capsule containing a gram of feces collected from a patient with clinical dysentery and containing large numbers of living bacilli.

Few are the human beings who do not suffer from the common cold once or several times every year, and few are the epidemiologists and laymen who do not believe that the affliction spreads readily by contact and that certain environmental circumstances, such as wet and cold weather, draughts, and fatigue, increase susceptibility to it. Since men find it so difficult to avoid "catching" a cold although they are most reluctant to "take" it, one could expect that it would be easy for the experimenters to "give" the disease to human volunteers willing to submit themselves to all the conditions assumed to be favorable for its transmission. But this has not proved to be the case. It has been known for almost two decades that one can elicit mild symptoms of coryza in a certain percentage of human beings by administering to them filtrates of nasal washings and discharges from individuals in the early stages of the common cold. Thus, the etiological agent appears to be a filterable virus. It has also been found in early experiments that human beings are less likely to develop symptoms when exposed to the infective dose in an environment conducive to physiological and mental well being.

Experimentation seems to confirm what had been suspected from observations in daily life, namely, that man is highly receptive to the common cold agent under special circumstances. But these circumstances are not easy to define as was the experience of the workers of the Common Cold Commission in England. In experiment after experiment, they introduced, intranasally into human volunteers large volumes of presumably infective discharges freshly collected from patients in the acute phase of the disease, but only a small percentage of the would-be victims developed acute signs of cold, even if exposed to air draughts in wet socks for hours immediately after having received the infective dose. Whatever the technique of infection used and whatever the environmental circumstances, the common cold so difficult to escape in ordinary life manifested itself to the experienced investigators who tried to conjure it only in a small percentage of their human volunteers.

GERM THEORY LIMITATIONS

It is somewhat entertaining to muse over the fact that although all textbooks of bacteriology and infectious disease present the Henle-Koch postulates as the very bedrock which gives permanent basis and scientific dignity to the germ theory of disease, these postulates have hardly ever been satisfied in man. In reality, the faith that micro-organisms play the primary role in the causation of disease grew out of the forcefulness of Pasteur's and Koch's convictions, rather than out of the unimpeachability of the evidence which they presented.

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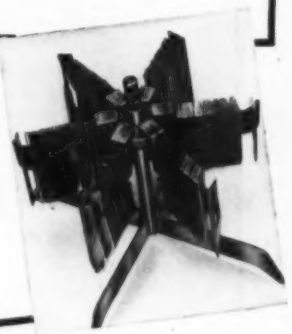

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Lest this statement be considered as a facile and irresponsible exercise in "debunking," let us admit immediately the obvious fact that Pasteur, Koch, and their disciples and followers based their conviction on facts of absolute validity. But these facts were valid only for the very limited and well-described circumstances under which they were observed. It was no small part of Pasteur's and Koch's vision and genius that they selected for the demonstration of their thesis experimental situations in which it was not only necessary but also sufficient to bring the host and parasite together in order to reproduce the disease. Farm and laboratory animals never carry the anthrax bacillus; all guinea pigs are susceptible to tuberculosis; injection of rabies virus under the dura of dogs always gives rise to paralytic symptoms and so forth. Thus, by the skillful selection of their experimental models, Pasteur and Koch could eliminate from their studies the factors other than the parasite which are necessary to demonstrate that infection can result in symptoms and pathological alterations. The techniques of experimentation which they worked out were designed to test the pathogenic properties of parasites under reproducible conditions—ideally a highly susceptible animal placed in a simple, defined, and constant environment. Useful as this artificial system has been for the study of some of the properties of microparasites, it has led to the neglect, and indeed has often delayed recognition, of the many other facts which are essential to the causation of disease under circumstances prevailing in the natural world—the physiological characteristics of the host and the physicochemical as well as social environment.

DISEASE COMPLEX IN ALL LIFE FORMS

The examples used so far have been taken from human pathology to illustrate the view that the causation of disease under natural conditions often involves the simultaneous operation of several independent etiological factors. In reality, however, this holds true as well for many varied types of pathological processes in all classes of living things.

In nature, many bacterial species called "lysogenic" carry in an inactive form (prophage) one or several bacteriophages potentially capable of causing their lysis. Under ordinary conditions, the prophage is apparently reproduced with each bacterial division without causing any detectable disturbance in the cell. This equilibrium can be upset by a number of nonspecific procedures—for example, irradiation of the lysogenic culture—in such a manner that the prophage is converted into active bacteriophage, multiplies abundantly, and causes the destruction of its host cell. Thus, the prophage behaves as a pathogen for the cell that carries it only when the latter is subjected to the proper kind of stimulus. One might say that the prophage makes the bacterium sensitive to the radiation, or that the radiation makes it susceptible to the phage, or that both agents are required for the causation of lysis. In fact, it has been shown that, in certain cases at least, the activating effect of radiation can take place only in media of certain composition, thus making even more complex the etiological determination of lysis.

As is well known, the agent responsible for mammary carcinoma among breeding female mice has all the characteristics of a virus. Although the Bittner virus occurs throughout the tissues of infected animals, it causes no obvious sign of disease and in particular no tumors until lactation has begun. Male mice do not develop breast cancer even if they carry or have ingested the virus, nor do female mice unless under the stimulus of continuous reproductive activity. Thus, whereas the virus can remain latent in the animals which harbor it, the hormonal and other stimuli which accompany reproduction and lactation are necessary and sufficient to induce it to elicit pathological reactions. In other words, these physiological stimuli are, as much as the virus, etiological agents of the mammary tumors of mice.

All the pathological processes so far mentioned to illustrate the concept of multiple etiology involve the participation of micro-organisms or of viruslike agents. But one could also find in other fields of pathology many examples in which the causation of disease probably

requires that several independent factors act together or in succession. Thus, it is well recognized that conditions as varied as hemorrhagic shock, peptic ulcers, or degenerative diseases are multifactorial phenomena requiring for their genesis the simultaneous occurrence of several physicochemical and vascular disturbances conditioned by a variety of organic and psychogenic controls.

The problem of causation of the plant cancer, known as crown gall, because of the precise knowledge which has been gained of its physiological determinants and chemical basis, illustrates in a striking manner the conceptual difficulties involved in the determination of etiology. It is possible to induce at will characteristic tumors (crown galls) by inoculating sunflower or certain other plants with pure cultures of *Agrobacterium tumefaciens*. Since no other foreign organism or substance is known to be capable of causing this pathological reaction, it seems fair to regard *A. tumefaciens* as the specific etiological agent. It has been established, however, that many of the secondary tumors developing on the same plant at sites removed from the initial infection are free of bacteria, and yet can be transferred in series to new plants, or propagated in tissue culture as self-reproducing structures. It is possible also to eliminate the bacterium from the tumor tissue by controlled heating, without affecting the power of autonomous growth of the latter. Thus, production of the cancer can be made independent of *A. tumefaciens*, which was at first its essential etiological agent.

It is known furthermore that extensive invasion of the plant by the bacterium may take place without resulting in tumor formation. Only plant cells which have been conditioned by certain stimuli associated with wound healing are rendered susceptible to transformation into tumor tissue by the bacterium. The physiological state of the host cells should, therefore, be considered also an etiological determinant of crown gall.

Finally, it can be shown that whereas the normal plant tissue requires indole acetic acid and the cocoanut-milk factor for growth in tissue culture, the self-reproducing tumor tissue can synthesize these essential factors (and perhaps others) and therefore does not need them. It is this biochemical characteristic which permits it to grow profusely and in a completely uncoordinated manner. Thus, at the present state of the analysis, the biochemical etiology of the disease appears to reside in an increased synthetic power, but on the other hand, it takes *A. tumefaciens* originally to induce the change.

Depending upon the specialized interests of the investigator and the techniques that he chooses to use, the primary etiology of crown gall can be regarded as a specific bacterium, a transmissible cellular change dependent upon a certain physiological state of the cell, or a biochemical disorder. The problem of etiology can be studied at different levels by the bacteriologist, the oncologist, the physiologist, or the biochemist. All points of view are justified, and discoveries at any one of the levels of investigation can add to the understanding of the mechanisms of the disease and probably lead to some technique of control.

BIOLOGICAL AND ENVIRONMENTAL EQUILIBRIUM

Obviously the "normal" existence and performance of any living organism demands a state of subtle equilibrium between its different component parts, as well as between them and the factors—living and physiochemical—of the environment. Hippocrates referred to the ideal state of nature as a "universal sympathy." Biological equilibrium, being the outcome of evolutionary forces, is maintained only if nothing changes in the environment. Any change is likely to disturb the balance of forces upon which depends the maintenance of the normal state. "Disease," Virchow said, "is life under altered conditions."

The factor which disturbs the equilibrium may affect only one or a few individuals and give rise to isolated cases of clinical disease, or it may affect simultaneously many members of the community and cause crowd diseases.

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1. Cass, L. J. and Frederik, W. S.: Malt Soup Extract as a Bowel Content Modifier in Geriatric Constipation, Journal-Lancet, 73:414 (Oct.) 1953.

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and economic factors is sufficient proof that here again, as for infectious diseases, it is necessary to broaden the concept of etiology if one is to achieve a comprehensive picture of pathogenesis.

It is because the physician must deal with situations involving so many independent variables that clinical medicine has remained an art even today—an art based on wisdom and skill derived from experience as much as on scientific knowledge and reasoning. The skill symbolized by the gold-headed cane was not mere charlatanism. Experience had taught the good physician to manipulate many of the factors—physiological, psychological, and social—which affected the reactions of his patient.

But knowledge based on empirical experience either remains static or at best develops slowly. Progress demands systematic experimentation. Methodologically, there is danger in adopting too broad a biological and clinical point of view in the study of disease—the danger of substituting meaningless generalities and poor philosophy for the concreteness of exact knowledge. It is one of the most important contributions of the doctrine of specific etiology that it saved the science and practice of medicine from the morass of loose words and concepts. But insistence on concrete facts need not deter one from acknowledging that, under natural conditions, the etiology of most diseases is multifactorial rather than specific. The laboratory worker can always circumvent the difficulty resulting from the multiplicity of etiological determinants by devising operations that permit him to study each of these separately and in various combinations.

By using a broadened concept of etiology, encompassing intrinsic and extrinsic determinants of disease, he will eventually develop a therapeutic science that will incorporate the human wisdom and empirical skill of the traditional medical art. "The variable composition of man's body hath made it an Instrument easy to distemper . . ." Francis Bacon wrote. "The Office of Medicine is but to tune this curious Harp of man's body and to reduce it to Harmony."

MARRIAGE RATE DOWN SLIGHTLY IN 1953*

About 1,553,000 marriages took place in the United States during 1953. Although this was 20,000 more than the number in the preceding year, the marriage *rate* fell off slightly. In 1953, the rate was 9.7 per 1,000 population (including the armed forces overseas), compared with 9.8 per 1,000 in 1952. In both years, it was about two fifths below the level for 1946, when the rate rose sharply to the all-time high of 16.2 per 1,000.

The recent marriage rate is substantially above that of the early 1930's, even though it is at the lowest point in two decades. The reasons for the falling off in marriages in the two periods are quite different, however. At the beginning of the 1930's, unemployment and economic depression discouraged men and women from undertaking the responsibilities of family life. During the past few years, when business conditions have been remarkably good, the marriage rate has been relatively low because the number of single persons was depleted by the marriage boom immediately after World War II. In 1946 alone there were about 2,291,000 marriages in our country, and the total was not much below two million in each of the two years following. Another factor contributing to keep the current rate down is the smaller number of people "coming of age," as a result of the decline in births during the 1930's.

In most areas of the United States the number of marriages in 1953 showed relatively little change from the preceding year. Several States, however, experienced substantial changes. The number of marriages increased by about one sixth in Missouri and Delaware, and by more than one tenth in five other States—Vermont, North Dakota, Georgia, Oklahoma, and Nevada.

On the other hand, 14 States and the District of Columbia reported fewer marriages in 1953 than in the year before. In all but one of these—Arkansas

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, December, 1953.

—the decreases were less than 6 percent. In Arkansas, however, marriages fell off more than two fifths, due to the enactment of legislation requiring a premarital serological test. That State had been registering a steady decline in marriages during the early months of 1953, but the downswing was greatly accelerated after July 1st when the new law became effective. It is likely that many marriages which would previously have taken place in Arkansas are now occurring in adjacent States. No doubt, this shift accounts in considerable measure for the upswing in marriages experienced by Missouri, Mississippi, and Oklahoma.

In a majority of the leading cities, the number of marriages changed but little from that of the preceding year. In cities with at least 100,000 population, as a group, there were 1.3 percent more marriages in the first 10 months of 1953 than in the corresponding period of 1952. Fifty-six, or somewhat more than one half of these urban areas recorded increases; in 47 there were decreases. Only one large city—Birmingham, Ala.—reported a decline of more than 10 percent. By contrast, five cities reported increases of this magnitude—namely, Tulsa, Okla. (17 percent), Kansas City, Mo. (16 percent), Flint, Mich. (14 percent), Little Rock, Ark. (13 percent), and Memphis, Tenn. (11 percent). The upswing in marriages in Little Rock is particularly noteworthy, in view of the marked decline in the rest of the State.

In four of our five cities with populations exceeding 1,000,000, the rise in marriages from 1952 to 1953 was somewhat larger than that for the country as a whole. In the fifth, New York City, the number of marriages was practically at the same level in the two years.

LARGE POPULATION GAIN IN 1953*

The population of the United States increased 2,700,000 during 1953, bringing the total, including the armed forces overseas, to about 161,200,000 at the year's end. This large increment, exceeding somewhat the previous high gain registered in 1952, reflected not only a record-breaking number of births, but also a low death rate and a moderate increase from immigration.

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, December, 1953.

The new high record for births was not far short of the 4 million mark. The actual number, including an allowance for unregistered births, was more than 3,950,000, or nearly 25 per 1,000 population. The births in 1953 were nearly 2 percent greater than in the preceding year and more than 50 percent higher than in 1940. The year just ended was the seventh in succession in which births exceeded 3½ millions. In the past decade, the number of babies born reached

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The Southwest and the Rocky Mountain areas experienced the largest relative rise in births in 1953. In other sections of the country, Florida, Delaware, and Missouri showed increases well above the national average.

Infant mortality declined in 1953, establishing a new low estimated at 28 per 1,000 live births, or slightly below the previous minimum recorded the year before. The infant mortality rate in the year just ended represented a decline of 30 percent from that a decade ago and of more than 50 percent from that two decades ago.

The general mortality in the United States was also at a low level. The 1953 death rate was 9.6 per 1,000, or about the same as in the preceding year, which established a new minimum. This record is especially impressive because 1953 was marked by an outbreak of respiratory disease early in the year and a severe hot spell in late summer, both of which brought a rise in mortality over the seasonal normal. The death rates at ages up to 55 were as low as or lower than those in any past year. When the final figures are in hand, and allowance is made for the increase in proportion of older people, the adjusted death rate for all ages combined may set a new low record.

The natural increase in population—the excess of births over deaths—reached a new high of about 2,425,000 in 1953. This was somewhat above the previous maximum recorded the year before, and more than double the annual increase during the 1930's.

The rapid rate of population growth which began in 1946 has now continued for eight consecutive years. The annual gain in population exceeded 2½ millions in all but one of these years. During this eight-year period, the population grew by more than 20 millions; just prior to 1946, it took 17 years to produce such an increase. The magnitude of the recent population gain in our country can be better appreciated when one realizes that it exceeds by more than one third the present population of Canada.

Every age group has contributed to the growth in population, with preschool children showing the largest gain. Thus, as against a rise of one fifth in the total population since 1940, the number of children under 5 has increased by about two thirds. These youngsters now constitute 11 percent of the population, compared with 8 percent in 1940. At the same time, the elders in our country have also grown rapidly both in number and in proportion to the total. Since 1940, the people at ages 65 and over have increased by nearly 50 percent and now comprise about 13½ millions, or more than 8 percent of the population. Those at the main working ages of life have also experienced a substantial increase in numbers although they have decreased slightly as a proportion of the total population.

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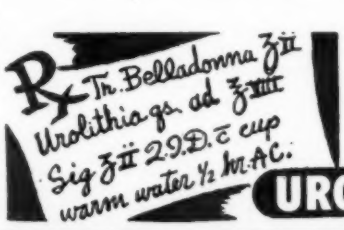

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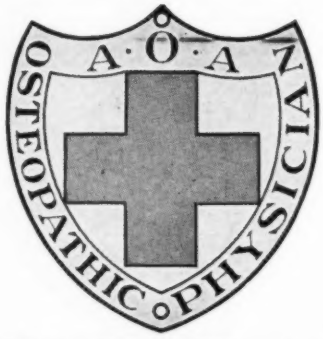

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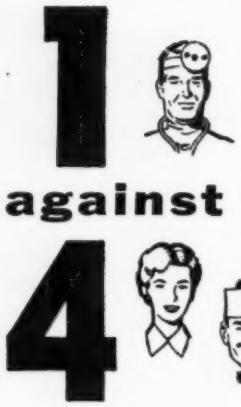

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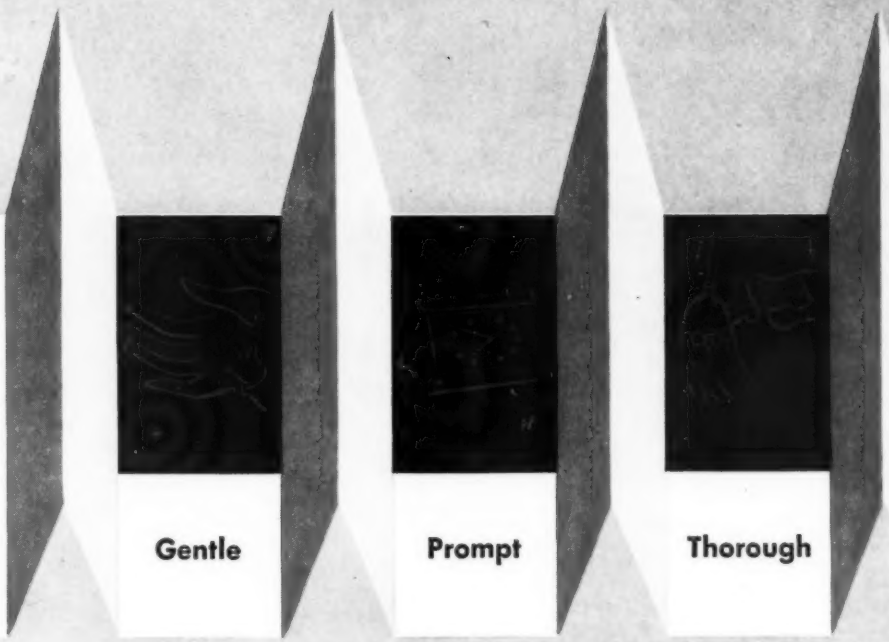

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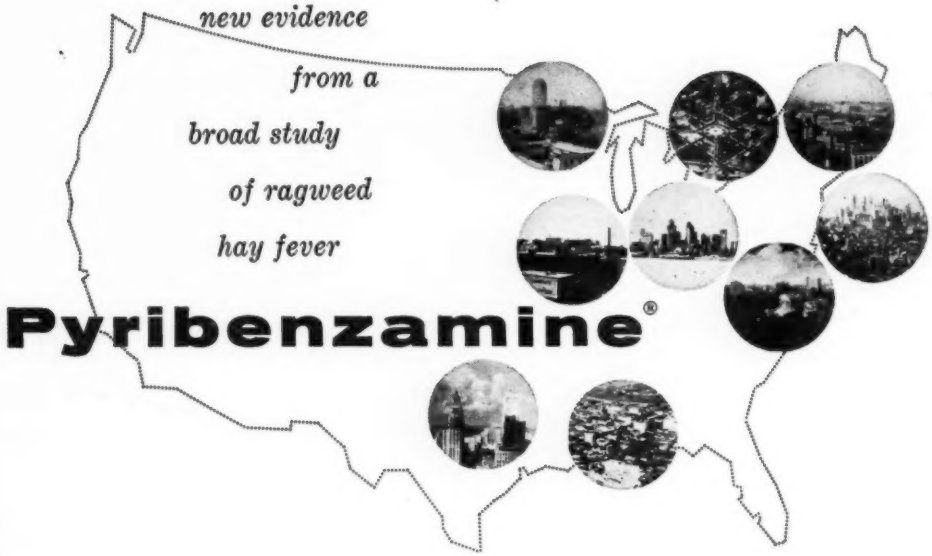

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